This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.

+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.

+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.

+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`

LANE MEDICAL LIBRARY STANFORD
L341 .O62m 1900 STOR
Diseases of the nervous system : a text-
24503404907

LANE
MEDICAL LIBRARY
SEAL OF THE LELAND STANFORD JUNIOR UNIVERSITY
1885

Dr. Donald Smith

DISEASES

OF THE

NERVOUS SYSTEM

LANE MEDICAL LIBRARY
STANFORD UNIVERSITY
MEDICAL CENTER
STANFORD, CALIF. 94305

A TEXT-BOOK FOR STUDENTS AND PRACTITIONERS OF MEDICINE

BY

H. OPPENHEIM, M.D.

PROFESSOR AT THE UNIVERSITY OF BERLIN

AUTHORIZED TRANSLATION
BY

EDWARD E. MAYER, A.M., M.D.

PITTSBURG, PA.

FIRST AMERICAN
FROM THE SECOND REVISED AND ENLARGED
GERMAN EDITION

WITH TWO HUNDRED AND NINETY-THREE ILLUSTRATIONS

LANE MEDICAL LIBRARY
6749
SAN FRANCISCO

PHILADELPHIA AND LONDON
J. B. LIPPINCOTT COMPANY
1900

Copyright, 1900,
by
J. B. Lippincott Company.

Electrotyped and Printed by J. B. Lippincott Company, Philadelphia, U. S. A.

2 m
0

TO THE MEMORY
OF
CARL WESTPHAL

PREFACE TO THE SECOND EDITION.

THE recognition given to this book in its original form, both by general practitioners and by the most illustrious specialists in nervous diseases, has prompted me in this new edition to make only such changes as have become necessary by reason of the advancement of the science of which it treats and also because of my own enlarged experience. Although the specific presentation of individual diseases has been strengthened by the addition of new matter and by greater thoroughness of treatment, the book in its fundamental plan in no way differs from the first edition. In the revision I have departed from only one of my original principles,—namely, in having, in this second edition, considerably augmented the literary references. I must say, further, now that the revision is complete, that I am doubtful whether this addition is of much advantage.

The number of illustrations has been largely increased, and, although some have not been reproduced as faithfully as I would have desired, I hope that they will serve to aid in the understanding of the diseases and their anatomical foundations. Where no acknowledgment is made that the figures have been taken from other works, it is to be understood that they are from my own observations and preparations.

The publishers are to be congratulated upon the fact that, although much enlarged as regards the matter presented, this new edition has not to any extent increased in size. I desire to give special acknowledgment and thanks to my assistants, Drs. Cassirer and G. Flatau, for the preparation of the photographs and the reading of the proofs.

THE AUTHOR.

PREFACE TO THE FIRST EDITION.

IN offering this work to the present and future students of nervous diseases, I feel the responsibility which such an undertaking involves. I have tried to present, from the vast amount of literature on the subject, only that which appears to have a sure basis and is founded on facts. In addition to the inadequacies and defects in our knowledge of nervous diseases, the limits and compass of my own experience have compelled me to add much which is not yet accurately known.

My endeavors were, above all, to make this book useful to practitioners. Much stress has, therefore, been laid upon symptomatology, diagnosis, prognosis, and therapeutics, while only so much space was given to pathological anatomy as was needed to shed light upon the course of a disease or to pave the way for a diagnosis. The normal anatomy and physiology of the nervous system have been considered in a concise yet complete manner, and their exposition has been enriched by appropriate plates. Literary references have been omitted, as they would have made the book larger than I wished it to be. Only in the more important investigations have names been cited. In doing this I am conscious that I may be severely censured.

The specialist will notice that I have freely consulted the extant text-books and hand-books of nervous diseases, and especially monographs, which form the chief source of our knowledge; he can, however, not help remarking that all that I have written is corroborated by personal observation and knowledge.

The most difficult part of my task was in the presentation of the therapeutics of nervous diseases. In the sections devoted to treatment I make free use of everything that has been recommended by our leaders in nervous therapeutics as well as of what has been tried by myself, and hope that I have neither been too sceptical nor fallen into the more dangerous error of being too free and uncritical.

At the moment of sending this work to press, I owe it to myself to express my thanks to those whom, in a certain sense, I look upon as collaborators. When, soon after the death of my teacher, Westphal, I forsook, after many years of labor, a field of work that had become endeared to me, and confined myself to polyclinic duties, it was the staff-physicians of one of our hospitals who enabled me to pursue my clinical and anatomical work. I am, therefore, deeply indebted to Professors Ewald and Langenbuch and Dr. Rotter, and especially Professor Dr. Moses, who placed at my disposal the extensive material of the city hospital.

I owe thanks also to Miss von Mayer and to Mr. Krause and Dr. Kroug who, among others, have assisted in the preparation of the photographs and illustrations.

H. Oppenheim.

TRANSLATOR'S PREFACE.

Professor Oppenheim's work has deservedly acquired exceptional popularity on the Continent. The translator hopes that it will in its present form appeal to the English-reading profession, thereby justifying the translation. Only minor alterations in the text have been made, and some of the illustrations have been altered to render them suitable to an English work.

CONTENTS.

I. GENERAL PART.

Methods of Examination; General Symptomatology; Objective Examination.

PAGE

The Anamnesis 15
Examination of the Mental State 17
The Muscular System; Examination of the Motility 19
Examination of the Tendon Reflexes 20
Examination of the Active Movements 24
Muscular Functions: Muscles of the Shoulder and Arm 25
The Muscles of the Pelvis and of the Lower Limbs 31
The Muscles of the Head and Vertebræ 35
Disturbances of Co-ordination 37
Tremor 39
The Electrical Examination 41
Examination of the Mechanical Irritability of the Muscles and Nerves.... 52
Examination of the Gait; Disturbances of Walking 52
Examination of the Sensibility 54
Disorders of Sensation 59
Skin-Reflexes 64
Spasms (Hyperkinesis) 67
Vasomotor, Secretory, and Trophic Disorders 68
Examination of the Special Senses; Disorders of Function of the Special Senses 72
Examination of the Remaining Cranial Nerves: Muscles of the Eyeball.... 77
The Sensibility of the Face 84
The Masticatory Muscles (Trigeminal Nerve) 85
The Muscles of the Tongue (Hypoglossal Nerve) 85
The Muscles of the Palate and Pharynx. Deglutition and its Disorders..... 86
The Muscles of the Larynx. Disturbances of their Functions 88
Disorders of Speech 90

II. SPECIAL PART.

SECTION I.

Diseases of the Spinal Cord.

Anatomy, Physiology, and General Pathology of the Spinal Cord 91
Secondary Degeneration 110
Spinal Localization 114
Unilateral Lesions of the Spinal Cord; Brown-Séquard's Paralysis 120
The Arteries of the Spinal Cord 122
Classification of Diseases of the Spinal Cord 123

The Systemic Diseases of the Spinal Cord.

PAGE
Tabes Dorsalis (Locomotor Ataxia) 123
Spastic Spinal Paralysis (Lateral Sclerosis) 140
Congenital or Juvenile Spastic Pareses 143
Combined Diseases of the Posterior and Lateral Tracts 146
Hereditary Ataxia (Friedreich's Disease) 149
Acute Anterior Poliomyelitis; Acute Atrophic Spinal Paralysis; Spinal Infantile Paralysis 152
Acute Anterior Poliomyelitis of Adults 159
Subacute and Chronic Anterior Poliomyelitis 160
Amyotrophic Lateral Sclerosis 163
Progressive Muscular Atrophy 167
Spinal Type of Progressive Muscular Atrophy 167
Hereditary or Familial Types of Progressive Muscular Atrophy of Spinal Origin 171
Primary Myopathy; Progressive Muscular Dystrophy 171
Neurotic or Neural Type of Progressive Muscular Atrophy 177
Thomsen's Disease (Congenital Myotonia) 179

Diffuse or General Diseases of the Spinal Cord.

A. *Spinal Cord Affections due to Diseases of the Vertebræ.*

Luxations and Fractures of the Vertebræ 181
Caries of the Vertebræ; Tubercular Spondylitis 187
Carcinoma and other Tumors of the Vertebræ 192
Syphilitic Diseases of the Vertebræ 193
Arthritis Deformans of the Spinal Column 193

B. *Spinal Cord Diseases due to Meningeal Trouble.*

Spinal Meningitis 194
Chronic Spinal Meningitis 196
Pachymeningitis Cervicalis Hypertrophica (Hypertrophic Cervical Pachymeningitis) 196
Chronic Syphilitic Meningitis (Arachnitis Gummosa, Syphilitic Meningomyelitis, etc.), and other Spinal Cord Diseases of Syphilitic Origin 198

C. *Primary Diffuse Diseases of the Spinal Cord.*

Myelitis 204
Senile Paraplegia 209
Multiple Sclerosis 209
Abscess of the Spinal Cord 218
Hematomyelia; Apoplexia Medullæ Spinalis 219
Diseases of the Spinal Cord resulting from Pronounced Variation in Atmospheric Pressure; Diver's Disease; Caisson Disease 222
Tumors of the Spinal Cord 222
Spinal Gliosis and Syringomyelia 227
Diseases of the Cauda Equina (and the Filum Terminale) 233
Diseases of the Spinal Cord without any known Anatomical Lesion 235
Congenital Malformations of the Spinal Cord and its Membranes 236
Reflex Paralyses; Exhaustion Paralyses 237
Periodical Paralyses and Osteomalacious Paralyses 238

SECTION II.

Diseases of the Peripheral Nerves.

PAGE
Anatomy of the Peripheral Nerves.. 239

Peripheral Paralyses.

Traumatic Paralysis of the Peripheral Nerves.............................. 240
Neuritis (Inflammation of Nerves)....................................... 248

Peripheral Paralyses of Spinal Nerves.

Paralysis of the Phrenic Nerve.. 252

Paralysis of the Brachial Plexus; Combined Peripheral Paralysis of the Nerves of the Shoulder and Arm.

Erb's (Duchenne's) Shoulder Paralysis 255
Lower Plexus Paralysis (Klumpke).. 257
Total Plexus Paralysis.. 258
Obstetrical Paralysis 259

Paralysis of Individual Nerves of the Shoulder and Arm.

Paralysis of the Long Thoracic Nerve.................................... 260
Paralysis of the Circumflex Nerve; Paralysis of the Musculocutaneous Nerve 261
Paralysis of the Suprascapular Nerve 262
Paralysis of the Musculospiral Nerve..................................... 263
Paralysis of the Median Nerve 269
Paralysis of the Ulnar Nerve.. 271

Peripheral Paralysis of the Nerves of the Lower Extremity.

Paralyses of the Crural and Obturator Nerves, etc........................ 274

Peripheral Paralysis of the Cranial Nerves.

Paralysis of the Nerves of the Ocular Muscles............................ 279
Periodical Paralysis of the Oculomotor Nerve............................. 284
Paralysis of the Trigeminal Nerve.. 286
Facial Paralysis (Prosopoplegia) 291
Primary and Isolated Peripheral Diseases of the Auditory Nerve........... 299
Paralysis of the Glossopharyngeal Nerve.................................. 301
Paralysis of the Vagus Nerve.. 302
Paralysis of the Accessorius (Spinal Accessory) Nerve.................... 306
Paralysis of the Hypoglossal Nerve....................................... 307

Multiple Neuritis (Polyneuritis)................. 309

Lead Paralysis 315
Arsenical Paralysis 319
Diphtheritic Paralysis 321
Puerperal Neuritis; Diabetic Neuritis; Senile Neuritis.................... 325
Landry's Paralysis; Acute Ascending Paralysis 332
Acute and Chronic Polymyositis... 337
Occupation Palsies; Professional Pareses................................. 341

PAGE
The Neuralgias 342

Neuralgia of the Trigeminal (Tic Douloureux, Prosopalgia, etc.) 351
Occipital Neuralgia 357
Neuralgia of the Phrenic Nerve 359
Brachial Neuralgia 360
Intercostal Neuralgia 362
Neuralgia of the Mammæ; Mastodynia; Irritable Breast 365
Lumbar Neuralgia 366
Neuralgia Ischiadica; Sciatica 367
Pudendo-Hemorrhoidal Neuralgia 375
Coccygodynia 376
Visceral Neuralgia 376

Nerve Tumors 377

SECTION III.

Diseases of the Brain.

Anatomy and Physiology of the Brain. Cerebral Localization.

The Membranes of the Brain 380
Fissures and Lobes of the Cerebral Surface 383
The Relation of the Skull to the Cerebral Surface 386
Histology of the Brain Cortex 388
Localization in the Cortex; Cerebral Localization 391
Conducting Fibres and Tracts 399
Visual Tract 411
Central Ganglia 412
Nuclei of the Cranial Nerves 413
Vascular Supply of the Brain 424
Secondary Degeneration in the Cerebrum 428

General Symptomatology of Cerebral Diseases.

1. General Considerations 428
2. Focal Symptoms 433
   Motor Disturbances 433
   Disorders of Sensibility 443
   Disturbances of Vision 446
   Disorders of Speech 454

Diseases of the Cerebral Meninges.

Inflammations of the Dura Mater 467
Acute Inflammations of the Soft Cerebral Membranes 471
Acute (Purulent) Cerebral Meningitis or Leptomeningitis 471
Epidemic Cerebrospinal Meningitis 482
Tubercular Meningitis; Tubercular Basilar Meningitis 488
Chronic Meningitis 494

Disturbances of the Cranial Circulation 495

Cerebral Anemia 496
Cerebral Hyperemia 497

Diseases of the Brain Substance.

PAGE
Cerebral Hemorrhage 500
Softening of the Brain (Encephalomalacia) 517
Acute Encephalitis (Non-Purulent) 525
  1. Acute Hemorrhagic Encephalitis 526
  2. Acute Polioencephalitis 529
Infantile Cerebral Palsy 532
Abscess of the Brain 545
Thrombosis of the Intracranial Sinuses 556
Tumors of the Brain 564
Aneurisms of the Cerebral Arteries 583
The Parasites of the Brain 585
Hydrocephalus 589
Syphilitic Diseases of the Brain 597
Progressive Paralysis of the Insane (Dementia Paralytica) 611

Diseases of the Pons and Medulla Oblongata 623

Progressive Bulbar Paralysis 629
Infantile Hereditary Progressive Bulbar Paralysis 634
Acute Bulbar Paralysis (Apoplectic Form) 635
Acute Bulbar Neuritis 642
Compression Bulbar Paralysis 642
Pseudobulbar Paralysis and Cerebrobulbar Glossopharyngolabial Paralysis .. 644
Infantile Pseudobulbar Paralysis 647
Bulbar Paralysis without Anatomical Lesions 648
Ophthalmoplegia 652

Diseases of the Cerebellum 656

SECTION IV.

The Neuroses.

Hysteria 661
Hypnotism and Hypnosis 700
Neurasthenia 703
Morbid States which, as a rule, accompany Hysteria and Neurasthenia, but which occasionally may be regarded as Independent Diseases 725
Phobias 725
Imperative Ideas 726
Astasia—Abasia 730
Akinesia Algera 731
Traumatic Neuroses 733
Hemicrania (Migraine) 748
Headache (Cephalalgia) 756
Vertigo 759
Glossodynia 763
Epilepsy 764
Eclampsia 785
  1. Eclampsia Infantum 785
  2. Eclampsia Parturientium 786
Localized Muscular Spasms 787

PA

Localized Muscular Spasms.

Facial Spasm. Convulsive Tic 7
Masticatory Spasm 7
Spasms of the Hypoglossus, Glossospasm 7
Spasms of the Muscles of the Neck 7
Spasms of the Muscles of the Trunk and of the Extremities 8
Respiratory Spasm 8
Saltatory Reflex Spasm 8
Maladie des Tics Convulsifs. Tic Général 8
Paramyoclonus Multiplex (Friedreich); Myoclonia 8
Occupation Neuroses 8
Writer's Cramp; Graphospasm; Mogigraphia 8
Tetany 8
Chorea Minor (St. Vitus's Dance) 8
Chronic Progressive Chorea; Huntingdon's Disease; Hereditary Chorea 8
Chorea Electrica 8
Paralysis Agitans (Parkinson's Disease) 8

SECTION V.

Diseases of the Sympathetic Nervous System. Angioneuroses and Trophoneuroses.

Anatomy and Physiology 8
Angioneuroses and Trophoneuroses ....
Acute Circumscribed Edema of the Skin ....
Intermittent Articular Hydrops ....
Vasomotor Neuroses of the Extremities (Nothnagel); Acroparesthesia (Schultze) ....
Symmetrical Gangrene (Raynaud's Disease) ....
Erythromelalgia ....
Scleroderma ....
Progressive Facial Hemiatrophy ....
Basedow's Disease (Exophthalmic Goitre) ....
Myxedema ....
Acromegaly ....

SECTION VI.

Toxic Conditions with Predominating Nervous Symptoms.

Alcoholism ....
Morphinomania ....
Tetanus ....
Head Tetanus ....

DISEASES
OF
THE NERVOUS SYSTEM.

PART I.

GENERAL SECTION.

METHODS OF EXAMINATION. GENERAL SYMPTOMATOLOGY.

THE ANAMNESIS.

Owing to the fact that many points of aid in the diagnosis of a nervous affection are either ignored by the patient or not thought of sufficient importance to mention until his attention has been called to them, great care should be exercised in taking the history of a case. It is advisable for the beginner to preserve a fixed order in his examination; when experienced, it is not so necessary. We will concern ourselves here with general rules and procedures, taking up in order, first, family history; second, general etiology; third, previous history and onset of disease; fourth, subjective, and, fifth, objective symptoms.

The first inquiry should be in reference to *heredity*. A person is of a neuropathic diathesis whose ancestors or relatives have been or are afflicted with any nervous disease. When a patient complains of the same disease, we speak of direct; when affected with a dissimilar one, of indirect heredity. It is well to remember that a neuropathic tendency and a neuropathic diathesis are not the same, an abnormal weakness of the nervous system being possible from birth without any hereditary influence. The most important inherited diseases are the psychoses, epilepsy, hysteria, neurasthenia, and hemicrania. Morbid inclinations to suicide, alcoholism, etc., indicate a neuropathic diathesis. Chronic lead intoxication, gout, and tuberculosis all predispose to nervous affections, likewise consanguineous marriages.

The general etiology should be ascertained before the onset of the malady is sought after. Obtain exact knowledge as to whether spasms, loss of consciousness, headaches (notably migraine), vertigo, or gastric disorders occurred in any former period of the patient's history. acute and chronic infectious diseases are often causes of nervous troul which may not come on for months or years afterwards. Typh variola, diphtheria, scarlatina, and influenza are particularly fatal to nervous system. The influence of syphilis on, and the intimate relat ship of tuberculosis to, the nervous structures are well known.

A former history of alcoholism is of value as an etiological fa Other poisons, as morphine, cocaine, and chloral, are also harmful. occupation or calling gives opportunity for the absorption or inges of poisons, and it should be determined whether through this or in o ways lead, arsenic, mercury, copper, brass, or carbon disulphide l been taken into the system. *Sexual abuses* and other perverse ha should be inquired for. *Traumatisms, mental strains,* and *emotiona citements* of all kinds are of great importance in the etiology. Clin and seasonal influences should not be neglected.

After these inquiries have been finished, attention should be dire to the onset and subjective symptoms. It is well to remember that m nervous diseases develop in fits and starts,—*i.e.*, intervals of compara health may intervene between successive stages of the disease. The tient does not connect these periods; he may know that at some for time he had some eye or stomach trouble, but that it has the sligl connection with his present symptoms is not realized. For this re data concerning former symptoms can only be procured by careful q tioning. Much attention must be given to the subjective sympto The physical signs frequently in themselves reveal a lung or h disease. But the neurologist often must needs confine himself ex sively to the feelings and thoughts of the patient, a careful phy examination revealing nothing. This shows how closely one must how the patient feels, what sensations affect him, and what he has no concerning his condition. Time and patience are usually necessary, only thus can results be obtained.

It is self-evident that an examination directed to the nervous tem only is a faulty and incomplete one. The specialist is particul liable to fall into this error, and for that reason he should make it a to precede the examination of the nervous system by a general From neglect of this often arise blunders, and he is bound to suffer his carelessness. A close observation of the patient during the tal of the history frequently yields important results. The expression the countenance many times indicates the state of the mind—loss intelligence, apathy, indifference, emotion, etc. The demeanor during relation of his troubles, slight mannerisms, lively gesticulation, gen motor-restlessness, tremors or twitchings, rapid changes in the colo the face, sudden shrinking, as from imaginary terrors or thoughts,—all these are signs which are easily noticeable and which may serve as chief elements in the making of a diagnosis. The state of the blood and general nutrition, as far as can be gathered from flushing or pallidness of the skin and mucous membranes, should be investigated.

EXAMINATION OF THE MENTAL STATE.

The disturbances of the mind bear an important part in the symptomatology of nervous diseases. A knowledge of psychiatry must always accompany, if not precede, that of neurology. Here only some general points will be noticed. Frequent observations are necessary to obtain a knowledge of the *emotionality, imagination,* and *volition,* though at times gross disturbances, as found in the markedly degenerate, neurotic, and decadent, may be noticed at the first examination. In conversing with the patient, the examiner has opportunity to satisfy himself as to the thoughts and feelings, the condition of the memory, the ability for resolution and decision; also, the degree of moral discrimination and of auto-suggestive ideation must be studied at this time. It requires tact and ability as well as experience to make some patients communicative, and one must be careful not to confuse and silence them by careless, inept, and insistent questioning. Especially in the disclosures of delusions, hallucinations, and illusions great discrimination is necessary. As a rule, the subject should not be introduced directly, but gradually and indirectly during the course of a seemingly indifferent conversation. The confidence of the patient must first be secured. On the other hand, rapid and direct questioning may at times so arouse the patient as to procure ready and satisfactory responses.

The account of the sickness ordinarily serves as an occasion to discover the emotional nature of the patient as well as full particulars as to his disease. If this does not serve the purpose, questions as to his probable future and his ideas concerning the further progress of his disease often meet with success. In examining the *degree of intelligence,* the general culture and social status of the patient must be taken into account. Gross disturbances are apparent in ordinary conversation. The patient's story of his sickness already informs us concerning the condition of his memory. Ascertain whether he can remember events and can arrange and classify them. It is important to notice whether he can distinguish matters of the past from those of the present time. The patient may relate intelligently the occurrences of the last few days, and yet show marked lapse of memory as to the facts connected with his earlier years. It is likewise advisable to determine his ability to receive new thoughts and to reproduce them, to understand names, figures, etc.

It is especially judicious to find out if he can calculate well. Have him add and multiply, and watch not only for a correct solution, but with how much despatch the mental labor is done, as the time relations of mental acts are important. Let people of moderate culture give an account of their work and manners of living. Questions as to days and dates, age, marriage, number and names of children, etc., often reveal gross defects of the intelligence and memory. One repeatedly finds it necessary to interrogate friends and relatives concerning observed changes in the patient. It is particularly recommended that his writing be examined. Not only do some patients, from embarrassment, write more explicit and full accounts of their trouble than they give you verbally, but, on the other hand, a comparison of this with writing of a previous period frequently discloses changes in thoughts as well as in style and composition.

Examination of the Skull.—Inspection may show scars, exostoses, and the like. Developmental abnormalities are noticed by the practised eye at once. Still, it is always better to satisfy one's self by careful measurements concerning these relations. The largest circumference of the skull at the height of the external occipital protuberance and glabella is about fifty-six centimetres in adult males, and about fifty-four centimetres in adult females, between thirty-five and forty centimetres in the new-born, about forty-five centimetres at the end of the first year, and at the twelfth year about fifty centimetres. The naso-occipital arc, from the root of the nose to the occipital protuberance, is in men about thirty-five centimetres. Noticeable asymmetry of the skull, macro- or microcephalism, marked facial asymmetry, excessive jutting out of the maxillary region, especially the protruding of the lower teeth, are all signs of degeneration (stigmata hereditatis). To the somatic stigmata, in addition, belong the anomalies of the ear,—absence of its lobe, or excessive flattening of the same, absence of the helix or antihelix, or anomalies of them, large, protruding ears with depressed borders and shallow groove (Morel's ear), etc. Also harelip, cleft palate, the small, canoe-shaped, sunken palate, irregular teeth, retinitis pigmentosa, albinism, and many other anomalies, are looked upon as anatomical landmarks of a neuropathic diathesis. Too much importance, however, should not be given them, as almost all may occur in perfectly normal individuals.

The percussion of the skull likewise often reveals much in focal diseases of the brain and its membranes. Auscultation should not be forgotten, as abnormal murmurs occur in cephalic diseases more often than is generally accepted.

X-ray examination of the skull may yield important results, though as yet nothing positive has been discovered by its use except in a few cases.

THE MUSCULAR SYSTEM; EXAMINATION OF THE MOTILITY.

A considerable increase or diminution of the muscular volume, when it is confined to one side of the body or to one extremity, is recognized at first glance. Yet a conclusion based upon a comparison of symmetrical muscles and muscular groups of the two sides of the body, when only slight differences are present, may be a deceptive one. In such an objective examination both extremities should be placed in exactly the same position, and the muscles stretched or relaxed equally. It should be remembered that slight differences are found under normal relations, especially a preponderance of about one centimetre in circumference on the right side.

To determine the exact degree of emaciation, careful measurements with the tape-measure are necessary. Naturally, to further this, the extremities should be placed in similar positions. On the forearm and leg select those places where the measurements are the most important; in the upper arm, select the middle; in the thigh, start from a fixed point, about twelve to fifteen centimetres above the patella. One can measure best by placing the limbs in a flexed position, though they should be kept free from the surroundings, as by resting them on anything differences caused by disproportionate pressure may falsify the results. Measurements cannot be exact; small errors of one-half to one centimetre will always occur.

Muscular emaciation is detected by the presence of grooves and depressions in places which normally are filled out with muscular tissue. Undue increase of muscular substance or puffing out or knotting of a part thereof will betray muscular hypertrophy. It should not be forgotten that all the degenerative conditions of the muscles do not result in alterations of their volume; more often a normally appearing muscle may be far from healthy.

Palpation gives us uncertain data concerning the muscular condition. The degenerated muscles feel soft and flaccid; but, if there is a fibrinous degeneration, may be hard and compact to the touch. If the muscle is surrounded by or infiltrated with fat, it will feel doughy. Much experience and care is necessary in using palpation, and hasty decisions as to the condition of the muscles may lead to many mistakes. The excision of a small piece for microscopical examination is not advisable in practice, and even in scientific examinations it has lost much significance since the discovery that excision in itself may cause changes in the muscular tissue. The most exact and important method for the examination of the state of nutrition of the muscles is by *electricity.*

Muscular Tonicity.—Before proceeding to the examination of active movements, it is judicious to form an opinion concerning the tonicity of the muscular system, since alterations thereof greatly influence active motility. Muscular tone may be increased or decreased. We derive our information concerning its condition by examination of the *passive movements* and of the *deep* or *tendon reflexes.*

To institute passive movement, we grasp the extremity of the patient and endeavor to move it in every joint in all directions as far as the mechanical relations of the joints will allow. This is comparatively easy when there is no muscular rigidity to be overcome. One must be careful to see that the patient does not purposely or unwittingly contract his muscles, through awkwardness, from not knowing what is expected of him, or from anxiety and emotion. He is therefore asked to allow his extremity to be entirely supported by the examiner, and shown how this is to be done. If this is not sufficient, divert his attention from the examination by giving him some mental work, some figures to add, etc. Only when the passively raised leg falls from mere gravity when it is let go can this disturbing factor be considered removed and the further results of the examination be of any value.

Pathological rigidity of the muscles betrays itself (1) BY INCREASED DIFFICULTY IN PASSIVE MOVEMENTS. In severe cases every attempt at movement will show this. If you endeavor to abduct the thigh at the hip-joint, the tense adductors will stand out prominently, but the whole pelvis, not the thigh alone, is moved outward. A certain degree of force, often considerable, is necessary to overcome this rigidity, and as soon as this is accomplished the limb will return to its original position. Generally only a slight increase in muscular tonicity is found,—so slight that it is only noticeable when, from passive motion, it is increased through reflex action. In these cases slow passive movements can be instituted without any especial resistance. But as soon as an attempt is made to carry out brisk movements, then rigidity ensues. Especially is this the case in abduction of the thigh at the hip-joint and flexion of the leg at the knee-joint. It is especially noticeable upon the first few attempts; it decreases with each succeeding one, until further movements are not resisted.

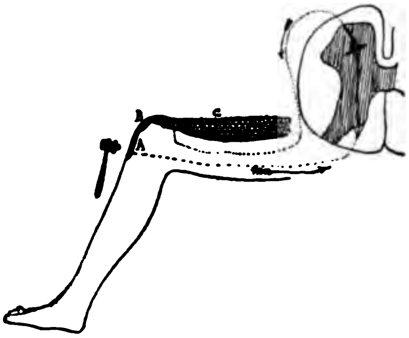
The increase of tonicity, muscular stiffness, rigidity, or the *spastic* muscular condition shows itself (2) BY THE INCREASED DEEP REFLEXES. On account of their intimate relationship with the muscular tonicity, it is desirable to follow the examination of passive motion by the examination of these reflexes.

THE EXAMINATION OF THE DEEP REFLEXES.—We understand by these—first written about by Erb and Westphal, by each unknown to the other—muscular contractions produced by mechanical stimulus given to one of the tendons. The most important one is the so-called *knee-jerk*, or patellar reflex. Always present in the healthy individual, its absence is of the greatest diagnostic importance.

Erb regards it as a reflex action. Westphal regards it as being a result of direct stimulation of the muscular substance, and that this stimulation produces a reflex action. Gowers believes that the contracted muscle stimulates the sensory nerve-endings, and that these by reflex action produce an increased irritability to mechanical stimulation (myotatic phenomenon).

To produce the knee-jerk the following procedure yields the best results. The leg of the reclining patient is bared, slightly flexed, and supported in the hock of the knee by the left hand of the examiner while the heel rests upon the couch or bed or table. The right hand meanwhile maps out the patellar tendon, providing it is not readily seen, and gives it a slight tap with the percussion hammer. Watch the upper thigh and see if the extensor cruris muscle contracts. This contraction, if strong enough, causes a jerk of the lower leg. Less attention should be paid to this than to the muscular contraction, as the former does not always occur. Do not confuse this muscular contraction with a simple vibration of the skin and muscular substance which results by contiguity from the effect of the tendon-blow. Providing the blow upon the tendon does not produce a contraction, ascertain whether an active rigidity (voluntary or involuntary) is not present. This is readily done by noting whether the leg, the supporting hand being removed, suddenly drops or not. Another method for testing the knee-jerk: The patient sits up and crosses one leg over the other, and is told to look upward. If in this position a tap upon the tendon does not produce a result, reinforcement by Jendrássik's method should be employed. Let the patient interlock his fingers and pull strongly, as if tearing them apart, but without separating them. If this is not successful, examine again with the patient sitting on the edge of the table or bed and his legs hanging loosely and freely. By pricking the patient with pins and needles, applying a strong light to his eyes, etc., you may not only engage his attention, but appear to strengthen the reflex. In rapid examinations the knee-jerk

FIG. 1.



Scheme of the patellar reflex-arc. (After Marie.)

may be tested by merely flexing his leg slightly while he is sitting in a chair. If the knee-jerk is found present, well and good, but its absence proves nothing, as then a new and accurate examination with the patient on his back must be instituted.

Increased knee-reflexes may be detected by the contractions which result from slight taps, even by mere touching with the finger, or—and what is an unmistakable sign—by a series of contractions of the quadriceps cruris. We rarely find complete rigidity of this muscle. Another way of demonstrating this clonus is the following: Grasp with thumbs and index-fingers the patella from above, push it quickly downward, and attempt with a slight resistance to keep it in this position. Clonic twitching of the muscle ensues, only ceasing when the patellar tendon is allowed to return to its normal position. This so-called *patellar clonus* is, however, an inconstant sign. The tendo Achillis can also produce a contraction. The knee should be slightly bent, the leg grasped with one hand, the toes forced gently upward with the other (without the patient helping the dorsal flexion), and the tendon struck a slight blow with the percussion hammer. The result is a plantar flexion of the foot.

This phenomenon, however, is not even constant in normal individuals. Its absence in itself, therefore, is not of pathological moment. Only when absent on one side, or when, during the period in which the patient is under observation, it is seen to disappear, is it of diagnostic importance.

The increase of this reflex is shown by the percussion hammer producing not a single contracture, but a clonic spasm, and is called

Ankle Clonus, or *Foot Clonus*.—This phenomenon may be more simply produced by tugging or pulling at the tendo Achillis. The knee is bent and supported by one hand, while the examiner grasps the foot with the other and presses upward. It is necessary to understand just how much force is needed to produce this clonus. Beginners generally use too much force, overflexing the foot. On the other hand, the pressure should not be removed too soon, but the tendon should be continually but gently stretched. With a considerable increase of the reflex, an active dorsal flexion acts generally as a stimulus to the clonus.

There is also a false ankle clonus which consists of a few twitchings at irregular intervals, and which seem to be a result of voluntary motion. It has been noted especially in hysteria. Another false form is found in paralysis agitans.

An increased muscular tonicity sometimes causes, on attempts to elicit the patellar reflex of *one* side, a contraction of the extensors on *both* sides, and even of some of the thigh muscles. It is also possible by percussion of the tibia to produce a contraction of the quadriceps and of the adductors of the thigh.

The intensity of the tendon phenomena varies considerably even in the healthy. Increase of single contractions is not of much importance, as it can be present in many physiological conditions; as, mental emotion, fear of the examination, and over-exertion. Every painful disorder, especially when in the legs, as sciatica, may produce an increased knee-jerk. In periostitis and rheumatism, phthisis, convalescence from acute fevers, and the itching exanthems the same condition is often found.

A mere increase of the deep reflexes, then, does not show a spastic condition. Marked rigidity as betrayed by resistance to passive movement is the important factor, the increase of the reflexes merely being a symptom that almost constantly accompanies it. Increased reflexes occur often without any muscular rigidity; we will see farther on that they occur in the different neuroses as a sign of increased excitation (hysteria, neurasthenia, etc.).

These phenomena take place also in the upper extremities. If you tap the styloid process of the radius with a percussion hammer a contraction of the supinator longus results, occasionally at the same time a slight contraction of the biceps. The arm in this examination should be held bent, between pronation and supination. In the same position a blow on the triceps will produce a twitching of this muscle. The tendon reflexes of the arm are, however, normally not very prominent, and may be absent, changeable, and weak. Only an increase or constant absence on one side of these reflexes is therefore of diagnostic importance. Increase of the reflexes of the arm are detected by a tap producing a strong contraction of more than one muscle or a clonic spasm of a muscle. A phenomenon analogous to the ankle clonus can be produced in the hand. Press the flexed finger-tips suddenly upward, forcibly hold them extended, and if clonus is present you will notice a clonic twitching of the flexors (*wrist clonus*). The masticatory muscles also present a reflex phenomenon,—the *mandibular reflex*. Place the finger or a spoon or something similar upon the lower teeth, tap it smartly, and a slight contraction results which raises the jaw. When the mere pulling down of the lower jaw causes a clonic spasm (*masseter clonus*) we have a spastic condition which is pathological.

A *decrease of muscular tonicity* (hypotony, atony) is not rare. It may be present normally, though generally accompanied by some pathological condition, as atrophy. Decrease in muscular tonicity, which is generally accompanied by a flaccid joint-capsule, is shown: (1) BY DECREASED RESISTANCE TO PASSIVE MOTION; (2) BY DECREASE OR ABSENCE OF THE DEEP REFLEXES. If a knee-jerk can not be obtained after trying all the methods outlined above, in all positions of the limb, with the attention concentrated on something else, and with Jendrássik's reinforcement, then we may truthfully claim that it is absent. However, before this result may be considered of clinical importance, we must decide whether a *mechanical* cause produces this result. An inflamed knee-joint, a dislocation of the patellar tendon, or an ununited fracture of the patella may cause such a condition. Even an abundance of fatty tissue which has deeply buried the tendon, or a considerable edema, can cause an absence of the knee-jerk. In some individuals the tendon is so short and lies so deep that the blow of the percussion hammer does not reach it. If it is very flaccid and deeply embedded, we may produce the reflex by first placing the leg in extreme flexion. In cachectic individuals I have often been forced to do this.

An absence of the knee-jerk is a sign of much importance. Although *Westphal* described this condition as taking place only in tabes dorsalis, yet it is so allied with his name that the absence of the knee-jerk has been, though badly, named "*Westphal's sign.*" It should not be forgotten that the knee-jerk may be absent in other conditions than diseases of the nervous system. Deep narcosis, all unconscious conditions, increased temperature, and exhaustion from over-exertion, may produce a loss of the reflexes.

It is very difficult to recognize a decreased knee-jerk. If with reinforcement it is only slightly noticeable, and the twitching is confined to only one part of the quadriceps, as, for instance, the vastus internus, we may safely say that a pathological decrease is present. Where only one side is affected, comparison with the sound side makes a decision easier.

Following the examination of the muscular tonicity, we take up the

EXAMINATION OF ACTIVE MOVEMENTS.

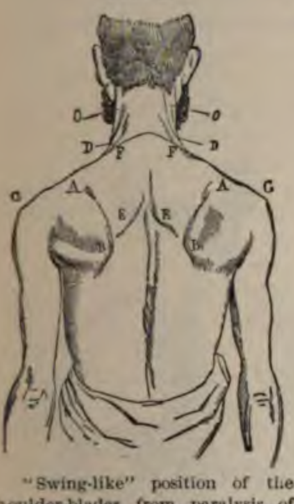
To proceed systematically, it is recommended to precede the examination of the complex movements with the simple ones. For example, examine the motility of the leg with the patient lying on his back, and then investigate his walking and running ability. In practice, however, we have formed certain conclusions concerning the gait from merely seeing the patient approaching and entering the room. Always first decide whether there is any mechanical resistance to ordinary movements. Notice especially whether joint-diseases with stiffness and atrophy of the fasciæ, shortening of the tendons or muscles through scars, etc., are present. Muscular rigidity often produces disturbance of motility. Pain may often cause lessened motion, which may simulate paralysis. In those persons, especially children, who are unable to acquaint you with the above facts, hasty examinations may cause grave errors.

The active mobility of the legs is next to be examined. Placing the patient upon his back, he is asked to move his extremities in all directions in maximum alacrity; to extend, flex, abduct, and adduct his leg, roll the hip-joint outward and inward, etc. By these movements the *limitations* and *quickness* of motion are ascertained. It is important to test the strength of the movements. The dynamometer can be dispensed with. One can judge the strength of the patient better by exerting resistance to every movement that you ask him to execute. Practice is essential to judge in this way the motor strength with any degree of accuracy. Test every different manner of movement and compare them with those of the opposite side of the body. It should be remembered that the left arm is generally weaker than the right (the relationship being about 4 to 5). Determine also whether all the muscles that should do so take part in a given movement. The *functions* of each and every muscle should be accurately known, as well as their position and appearance when paralyzed.

MUSCULAR FUNCTIONS.

Muscles of the Shoulder and Arm.—The *trapezius* muscles, acting together, raise the shoulders and bring the shoulder-blades towards the median line. Acting singly, the trapezius lifts the shoulder of the same side, draws the head backward, and at the same time turns it slightly towards the opposite side; for instance, the contraction of the right trapezius turns the chin towards the left.

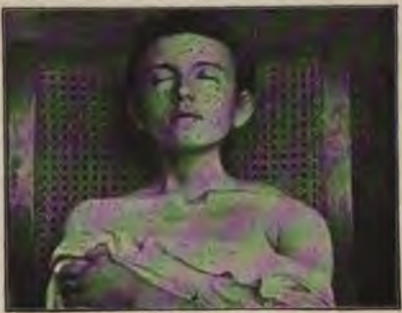
FIG. 2.



"Swing-like" position of the shoulder-blades from paralysis of the trapezius. (After Duchenne.) The levator anguli scapulæ is not paralyzed. The lower angle, B, B, approaches the vertebræ, the inner superior angle, A, A, is separated from it. The acromion, G, G, is depressed. D, D. levator anguli scapuli muscle; C, C, sternocleidomastoid.

The clavicular portion, which extends from the occiput to the outer third of the clavicle, moves the head in the above manner when the shoulders are fixed. It is called the respiratory portion because it aids in deep respiration. When paralyzed, the shoulder is immovable during breathing. The middle portion, which extends from the ligamentum nuchæ and the three upper dorsal vertebræ to the acromion and outer portion of the spine of the scapula, elevates the shoulder-blade. When well developed, a short neck is the result. In paralysis of this part the acromion sinks from the weight of the arm,

FIG. 3.



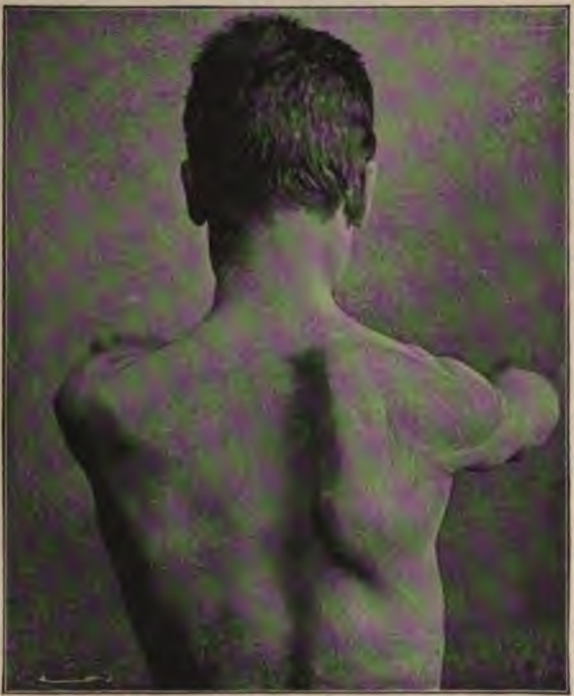
Improper position of the clavicles from paralysis of both trapezius muscles.

pectoralis major.) In order to detect the paralysis, we ask the patient to extend both arms forward and press his palms together. This, if it can be accomplished, is done with but little force.

The *latissimus dorsi* draws the elevated arm backward and downward, the depressed arm inward and backward; acting singly, it pulls the dorsal end of the trunk to one side, and extends it when acting conjointly.

The *teres major* adducts the arm towards the trunk, provided the shoulder-blade is fixed; draws the scapula outward when the depressed arm is fixed, and rotates it so that the acromion, and therefore the shoulder, is raised. Paralysis of it does not cause much disturbance. The long head of the triceps and the coracobrachialis belong to those muscles which support the head of the humerus in the capsule, and antagonize any subluxation of the humerus which would result from the action of the latissimus dorsi and the pectoralis major. If atrophied, when the arm is forcibly drawn downward the head of the humerus is subluxated downward; more so if at the same time a paralysis of the deltoid exists.

FIG. 7.



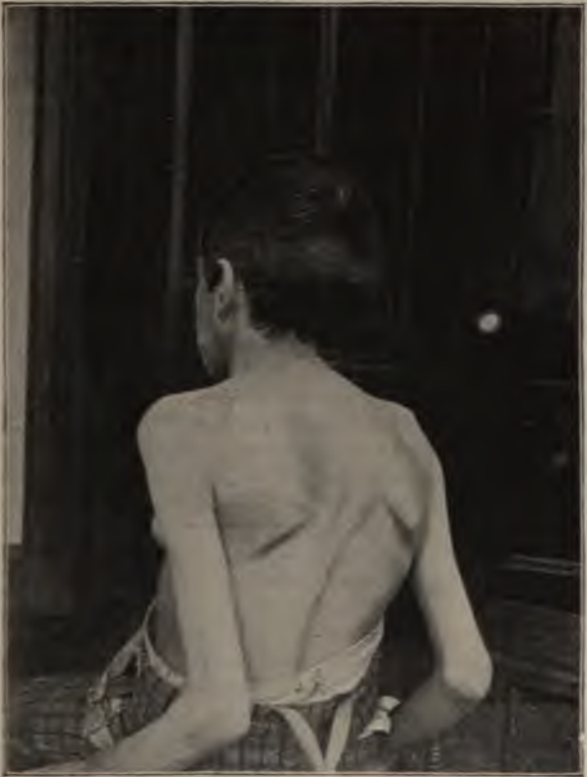
Position of the shoulder-blades in serratus-paralysis, when the arms are extended forward. "Angel-wing scapula."

The *brachial triceps* is the extensor of the forearm. If paralyzed, the arm can only be extended by its own weight. As soon as resistance is offered, or the arm is held vertically above the head, it is impossible to extend the forearm.

The *internal brachial* flexes the forearm without pronating or supinating it. The *brachial biceps* flexes and supinates at the same time, while the *supinator longus* slightly pronates and then flexes it. These muscles are equally contracted in forcible flexion. If, during flexion, pronation or supination occurs at the same time, one of these muscles is deficient. If all three are paralyzed, slight flexion may still occur, provided the flexors of the hand and fingers, which arise from the internal condyle of the humerus, and also the pronator teres, forcibly contract while the hand is fixed by their antagonists. The extensors of the hand and fingers can also promote flexion of the forearm after the latter is pronated and the wrist is fixed in an extended or flexed position. This artificial flexion, from the fact that it is impossible in the ordinary position of the hand and fingers, is easily recognized.

If the biceps alone is paralyzed, the forearm may be strongly flexed, but the patient tires easily and complains of pain in the shoulder. Paralysis of the supinator longus is recognized by the fact that in forcible flexion of the forearm against resistance the muscle does not show prominently. Its atrophy gives a spindle shape to the forearm.

FIG. 8.



Lateral curvature, showing projection of scapula, simulating "angel-wing" paralysis.

The *supinator brevis* supinates the hand with extended forearm. The pronator teres and the pronator quadratus are real pronators.

The *extensor carpi radialis longus* extends the hand and at the same time draws it towards the radial side; the *extensor carpi ulnaris* extends the hand and draws it towards the ulnar side. The *extensor carpi radialis brevis* alone is a simple extensor.

When all the extensors are paralyzed, the hand hangs by the side; and when passively raised, falls again to this position when let go. The palmar pressure is weak because the flexors of the fingers can only be brought into complete action in extension of the hand. If the hand is passively brought into extension, the palmar pressure is correspondingly increased.

The *extensor communis digitorum*, as well as the *extensor indicis* and *extensor minimi digiti*, extend the last phalanges and slightly separate the fingers from each other, abducting them from the middle finger.

In strong contraction of the extensor communis digitorum the hand, at the wrist-joint, is slightly extended. The extensor communis digitorum has nothing to do with the extension of the second and third phalanx.

The *flexor carpi radialis* flexes the hand and slightly pronates it, so that the palm is slightly turned ulnarward; the *palmaris longus* simply flexes the hand, while the *flexor carpi ulnaris* flexes especially the ulnar side of the hand and supinates the hand so that the palmar surface looks to the radial side. The fifth metacarpal bone is also flexed upon the carpal by this muscle. Paralysis of the flexors of the hand does not cause much anomaly of position, as the hand flexes by its own weight. If the flexors of the fingers are intact, they to a certain degree may assume the functions of the paralyzed flexors of the hand.

The *flexor sublimis digitorum* flexes the second, the *flexor profundus digitorum* the last two phalanges. They have nothing to do with flexion of the first phalanx, except in extreme extension or when the other phalanges, notwithstanding their contraction, remain extended, when they cause also a flexion of the first phalanx. The flexors act the stronger the greater the extension of the hand.

In paralysis of the *flexor sublimis digitorum* the second phalanx can from overweight of the extensors (interossei) gradually be flexed towards the first, and even subluxated; in paralysis of the flexor profundus this subluxation can happen between the second and third phalanges, yet is rarely seen, because an isolated paralysis of these muscles is uncommon.

It is important to know perfectly the functions of the *interossei* and *lumbricales*, because these are affected so often and in the most varied forms of disease. The external and internal interossei produce abduction and separation of the fingers. The movement is only complete when the hand is extended at the metacarpo-phalangeal joint. If, therefore, you desire to test this function when the extensor communis digitorum is paralyzed, you must extend the fingers passively, and with the hand supported on something (the hand of the examiner will do) you can proceed to have him adduct and abduct the fingers. Another important function of these muscles is *to flex the basal phalanges, and at the same time extend the second and third phalanges* of the fingers. In doing this they are assisted by the lumbricales.

In incomplete paralysis of these muscles the lateral movement of the fingers suffers. With increasing paresis the extension of both interphalangeal joints is hampered, and a characteristic deformity ensues. In normal persons the hand at best is slightly flexed at all joints; in paralysis of the interossei (and lumbricales) the basal phalanges are flexed, the middle ones more than the last. Finally, the antagonists (the extensor communis digitorum on the one hand and the long flexors of the fingers on the other) gain the ascendency and draw the first phalanx into extreme extension, while the second and third are held firmly flexed (*griffen-hand, claw-hand, main en griffe*). (Figs. 9 and 10)

The Muscles of the Thumb.—The *extensor longus pollicis* extends both phalanges of the thumb and draws the whole thumb backward. If paralyzed, the metacarpal bone of the thumb inclines forward, and the second phalanx is flexed against the first. It can, however, still be extended, provided the adductor and flexor brevis are brought into action, the metacarpal bone being kept flexed and adducted, the first phalanx being also flexed. Extension of the first and the second phalanx is not possible at the same time.

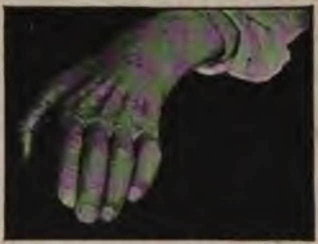
The *extensor brevis pollicis* is an abductor of the thumb; it brings the first metacarpal directly outward, extends the first phalanx, but has no influence over the second. Paralysis of this muscle is only of importance when the abductor longus pollicis is paralyzed synchronously. This latter also moves the metacarpal outward and at the same time forward,—*i.e.*, flexes it towards the wrist-joint, and with maximal contraction is a flexor of the hand. If the abductor longus pollicis and the extensor brevis pollicis are paralyzed, the thumb is adducted and a "volar hand" results.

The *flexor longus pollicis* flexes the second phalanx of the thumb. Its paralysis prevents this motion and hinders writing, etc.

Those muscles of the ball of the thumb which are inserted on the radial side of the first phalanx and metacarpal bone have the duty of moving the first metacarpal forward and backward, and of flexing the first phalanx and rotating it so that it is in apposition to the fingers.

The *opponens pollicis* merely moves the first metacarpal bone forward and inward, so that it remains directly opposite the second. The assistance of the abductor brevis and the outer part of the flexor brevis is necessary for the complete apposition of the wrist. In paralysis of all the muscles of the ball of the thumb the metacarpal bone of the thumb is brought into the same plane as the other metacarpals from the action of the extensor longus pollicis (ape-hand). (Fig. 11.) In paralysis of the abductor brevis and the opponens pollicis slight opposition through the flexor brevis is possible, but flexion of the first metacarpal is so incomplete that the thumb can only touch the tips of the other fingers when these are flexed at the interphalangeal joints.

FIG. 9.

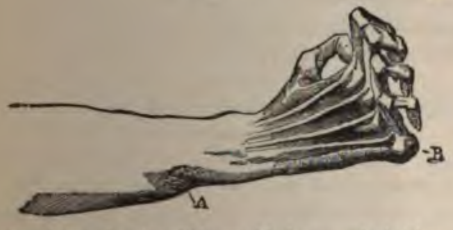


Partially developed "claw-hand" from atrophic paresis of the interossei lumbricales.

If the *adductor* is paralyzed, the first metacarpal is separated more than usual from the second and cannot approach it in a flexed position,—as, for instance, if the patient desires to hold a cane.

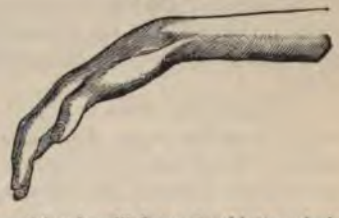
The Muscles of the Pelvis and Lower Extremity.—The *gluteus maximus* extends the thigh and slightly rotates it outward. With fixed limbs it extends the flexed trunk. This muscle acts especially in climbing stairs, running, arising from a chair, etc., and its paralysis hampers these movements. If, in paralysis of the glutei, the patient attempts to climb upon a chair, the pelvis inclines strongly forward. The *gluteus medius* is an abductor more than anything else. If the anterior portion contracts, the thigh is drawn forward and outward and at the same time rotates slightly inward; the posterior part draws the thigh backward and outward and rotates it out-

FIG. 10.



Complete "claw-hand" in an old ulnar paralysis (cicatrix at A). (After Duchenne.)

FIG. 11.



"Ape-hand" from atrophic (paralysis of the muscles of the ball of the thumb. (After Duchenne.)

ward. With fixed leg it draws the trunk to one side. The *gluteus minimus* has a similar action.

When these muscles are paralyzed, the leg cannot be abducted. In walking, the leg swings too far inward.

Especially noticeable is the excessive lifting and sinking of the pelvis in walking,—the *waddling gait.* The pelvis inclines to the opposite side from the paralysis in one-sided affections; when both sides are paralyzed, it inclines during walking to the side of the swinging leg.

The *pyriformis, gemelli, external* and *internal obturators,* as well as the *quadratus femoris,* rotate the upper thigh outward. If paralyzed, the leg is turned continuously inward.

The *ileo-psoas* flexes the leg at the hip-joint and turns the thigh slightly outward, the tensor fasciæ latæ exercising at the same time a slight rotation inward. If both flexors are paralyzed, walking is impossible; if paresis only exists, walking is hindered, and the thigh cannot be raised when in the reclining position the leg is held extended.

The *pectineus, adductores,* and *gracilis* adduct the thigh. The *pectineus* adducts and flexes at the same time. The *adductor longus and brevis* only slightly flex. All three cause a slight rotation of the leg outward. The *adductor magnus* draws the leg directly inward; its under portion rotates the leg inward.

Adduction of the leg is prevented by paralysis of the adductors. It also is turned outward from overweight of the abductors. In a paralysis which involves only the lower portion of the adductor magnus, adduction is accompanied by outward rotation.

The *quadriceps femoris* extends the lower leg. The rectus femoris is a strong flexor of the hips when the lower leg is extended. In paralysis of the extensors, standing is possible with extended knee; walking is also possible, but with difficulty. The limb must be kept extended, as an upright position would be impossible as soon as the flexors were brought into action. The stride is shortened to prevent flexion, as a long, swinging gait requires flexion of the knee.

Paresis of the quadriceps produces excessive flexion of the lower leg in walking; the difficulty of walking is decreased by the use of crutches or canes. To recognize paralysis of the quadriceps, extend the lower leg of the reclining patient, the thigh being flexed on the pelvis. The sole must not rest on anything. In sitting, the lower leg cannot be extended; and after being passively raised instantly drops again. Arising from a kneeling position is impossible, or in incomplete paralysis only possible by the patient placing his hands on his knees and pressing these backward.

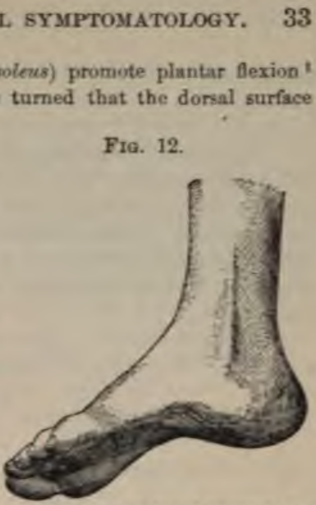
If the *vastus internus* is paralyzed alone, extension will draw the patella laterally by the action of the vastus externus. It may even result in luxation of the patella. Even in laceration of the ligamentum patellæ a slight extension of the lower leg is possible, by reason of the muscular fibres passing from the vasti to the sides of the tibia.

The *sartorius* causes flexion of the hip- and knee-joints and rotates the thigh outward; its action is an incomplete one. The *gracilis* flexes the lower leg slightly, adducts the leg more, and rotates it inward. The *biceps, semitendinosus,* and *semimembranosus* are flexors of the lower leg and extensors of the hip. They extend the hip-joint in ordinary walking (the glutei, etc., in climbing stairs). In paralysis of these muscles the pelvis would incline forward, if the patient did not instinctively by bending backward displace his centre of gravity backward. As the leg cannot be actively flexed any longer, the flexion is made easier by bending the hip-joint more than normally; the lower limb, by reason of its weight, then becomes bent. If the leg is stood upon, the quadriceps receives the most weight and the extension of the knee forces the leg into slight retroflexion. Walking, running, dancing, etc., are impossible.

The *popliteus* rotates the lower leg, when flexed, inward, and flexes it itself only slightly.

The *triceps suræ* (*gastrocnemius*, *plantaris*, and *soleus*) promote plantar flexion[1] and adduction of the foot. The foot also will be so turned that the dorsal surface looks outward. In simple plantar flexion the peroneus longus is also called into action. Its action is more forcible with extended knee (the gastrocnemius, which is inserted into the femur, slightly bends the knee); when the lower leg is bent upon the thigh, the soleus acts alone.

FIG. 12.

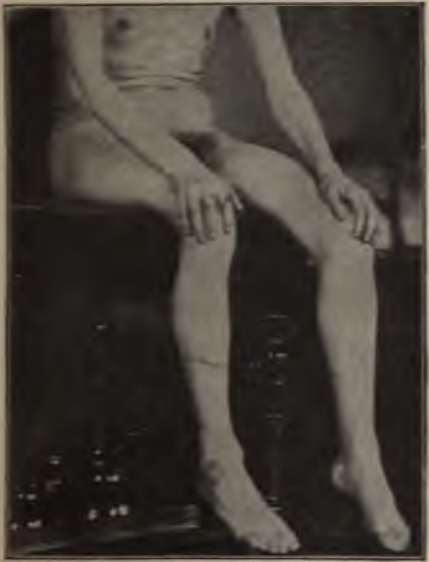


Pes valgus produced by secondary contraction of the peroneus longus muscle. Foot viewed from the outer side.

In paralysis of the triceps suræ flexion of the foot is almost impossible, and is never more than ninety degrees. The peroneus longus draws the first metatarsal downward, and causes valgus. (Fig. 12.) Gradually, through the extensors (the dorsal flexors), a hatchet-foot is produced. Contracture of the antagonists, however, does not always follow. The patient cannot raise himself upon his toes; walking is difficult for him. A secondary shortening of the muscles and fasciæ of the plantar region follows,—and, resulting therefrom, a marked arching of this region.

The *peroneus longus* has little to do with extension of the foot; it is especially an abductor. It depresses the inner and elevates the outer border, and draws the head of the first metatarsal downward and outward, thus decreasing the size of the anterior foot and increasing thereby the arch of the foot.

FIG. 13.



Position of the feet in paralysis of the extensors of the feet and toes, "foot-drop."

In paralysis of this muscle, extension of the foot takes place with adduction; the inner side of the front part of the foot is not supported any longer, and sinks from the pressure. In walking the foot only touches the floor with the outer border; the head of the first metacarpal is raised from the floor and the great toe is strongly flexed. The plantar arch decreases from standing, a flat-foot condition resulting. Walking is fatiguing; standing on the toes is not possible, or uncertain.

Pressure in walking upon the nerves of the plantar surface produces paresthesia and pain.

The *tibialis anticus*, *extensor longus digitorum*, and *extensor hallucis longus* produce extension of the foot. The tibialis anticus is at the same time an adductor; it draws the head of the first metatarsal upward and inward and lifts the inner border of the anterior part of the foot (at the same time the toes, especially the great toe, being flexed). The *extensor longus digitorum* extends the four toes slightly, but it is especially an extensor of the foot; it also lifts the outer border and abducts the foot. The extensor longus hallucis causes dorsal flexion of the first phalanx of the great toe, and at the same time aids in extension and adduction of the foot.

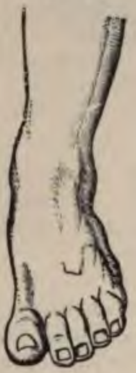
---

[1] Plantar flexion will be denoted as flexion, dorsal flexion as extension.

In paralysis of these muscles the foot cannot be raised; it hangs flaccid as soon as raised from the floor. (Fig. 13.) In walking the toes catch on the floor. To prevent this, the leg in walking is flexed at both hip and knee. The gait appears very peculiar for this reason (a beginner would regard it as ataxic). In chronic paralysis contracture of the flexors results, and from this *pes equinus* (pes equino-varus if the peroneus is paralyzed at the same time) is caused. This deformity gradually becomes a permanent one.

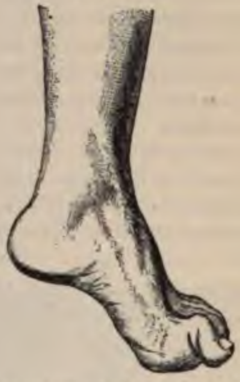
If only the *tibialis anticus* is paralyzed, extension of the foot is combined with abduction. (Fig. 14.) The long extensors of the toes, especially the extensor hallucis longus, are excessively stretched, the first phalanx of the large toe being chronically extended. (Fig. 15.)

FIG. 14.



Defective position of the foot from paralysis of the tibialis anticus muscle. The extensor communis digitorum attempts at extension bring the foot into a slight position of abduction. (After Duchenne.)

FIG. 15.



Pes equinus. Defective position from paralysis of the tibialis anticus. The tendon of the extensor hallucis longus is very prominent.

In isolated paralysis of the *extensor longus digitorum* dorsal flexion exists conjointly with adduction. The *peroneus brevis* abducts the foot and slightly raises its outer border, without flexing or extending it.

The *tibialis posticus* adducts the foot without extending or flexing it; at the same time the outer border is rendered convex and the head of the talus appears upon the back of the foot.

The paralysis of these muscles (the peroneus brevis and tibialis posticus) prevents simple adduction or abduction unaccompanied by flexion and extension, and produces, in time, deformity.

The functions of the foot are more severely harmed by loss of power of single muscles or muscle-groups than by paralysis of all the muscles of the foot, because in the latter case no extensive deformity occurs, but only a slight pes valgus is produced, since from the weight of the body the calcaneus is forced slightly outward. Inasmuch as for the development of the secondary contractures the erect posture is the criterion, this description does not apply to those who are bedridden. Provided the foot is fixed at a right angle to the leg by an appropriate surgical shoe, walking is possible.

The short extensor communis digitorum of the foot draws the toes farther dorsalward than does the long muscle of the same name.

The interossei pedis and the lumbricales produce not only abduction and adduction of the toes, but flex the first phalanx, while they extend the second and third.

The flexors digitorum (long and short), as well as the flexor hallucis longus, flex the last phalanges plantarward.

The *adductor*, *flexor brevis*, and *abductor hallucis* flex the first phalanx of the great toe and extend the second. The abductor and the inner head of the flexor brevis move the large toe inward, the adductor outward. These muscles contract in contraction of the foot preparatory to pushing it from the floor. If the extensors of the toes are paralyzed, the interossei become chronically contracted; the first phalanges are flexed, the last extended, thus producing an abnormal position of the toes. In paralysis of the interossei, the first phalanges are hyperextended and their heads subluxated, the second and third flexed (claw-foot). Walking is not impeded, but is painful. Walking and running are considerably influenced.

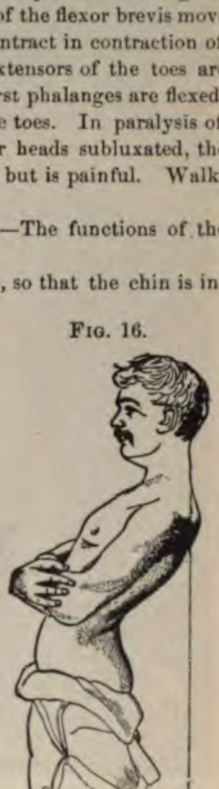
Muscles which move the Head and Vertebræ.—The functions of the following are especially noteworthy.

The *sternocleidomastoid* turns the face to the opposite side, so that the chin is inclined and raised on that side, while the head inclines to the same side as is the muscle, on which side the ear is at a lower level than on the other. If these muscles contract simultaneously, they bring the dorsally inclined head forward with elevation of the chin. If it is desired to test their function, have the patient recline, and then ask him to lift his head, at the same time resisting his efforts by pressure on the chin.

Their contour is so plainly evident under the skin that their contraction is easily seen; but do not rely upon this, as often they may be normally developed and yet not be seen.

Unilateral paralysis need not cause any abnormal position of the head, but generally the head is held in a position corresponding to the function of the muscle of the other side, and if kept there contracture may result. In paralysis on both sides, the head, which is strongly inclined backward, can be with difficulty bent forward.

FIG. 16.



Lordosis of the spinal column and defective posture due to paralysis of the extensors of the spinal column. (After Duchenne.)

The *recti capitis anticus* (*major* and *minor*) flex the head in the atlanto-occipital joint.

The *rectus capitis lateralis* bends the head to the side.

The *longus colli* is a flexor of the neck.

The *rectus capitis posticus* moves the head backward.

The *obliquus capitis inferior* or *major* rotates the head.

The *biventer cervicis* and *complexus major* draw the head backward.

The *splenius capitis et colli* draws the head backward and at the same time turns it towards the side of the contracted muscles.

The *sacrolumbar* and *longissimus dorsi* extend the lumbar and lower dorsal vertebræ. In unilateral action the vertebræ are drawn backward and to the side of the contracted muscles, so that the lower part of the spinal column as high as the eighth dorsal becomes twisted, and its convexity looks towards the opposite side.

The *semispinalis dorsi* and the *multifidus spinæ* are rotators of the vertebral column.

The *quadratus lumborum* bends the lower vertebral column towards the side.

If the erector trunci of both sides is paralyzed, the back in walking and standing is thrown backward so that a plumb-line from the third dorsal vertebra falls behind the os sacrum. (Fig. 16.) The pelvis is at the same time raised (action of the abdominal muscles).

There is present slight lordosis of the spinal column, which disappears on reclining. On sitting, the spinal column is arched convexly backward, and the patient prevents himself from falling forward by supporting himself with his hands.

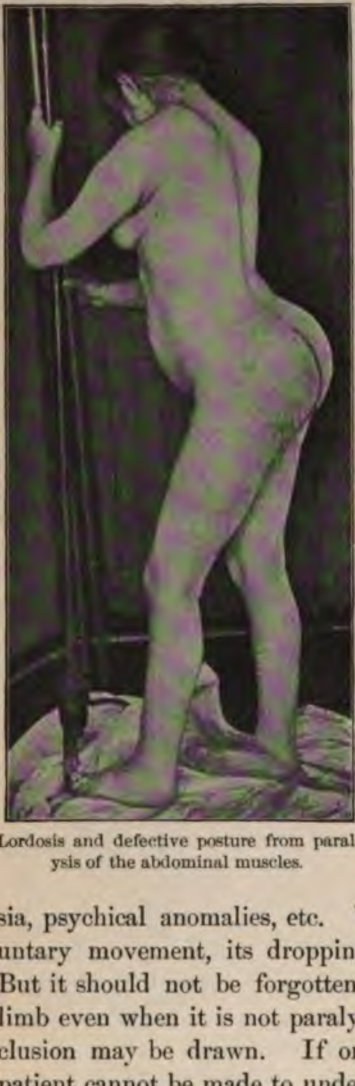
Paralysis of the abdominal muscles causes lordosis also, but a plumb-line from the dorsal vertebræ falls on the middle of the os sacrum, because the pelvis is strongly bent forward. The abdomen and nates show up prominently. (Fig. 17.)

It is only possible to rise from a reclining position by using the arms as a support.

Paralysis of the abdominal muscles also influences respiration, especially forced expirations, as in coughing, singing, and screaming, which are no more possible. The passing of the stools and urine is also hampered.

"Inasmuch as the viscera which push the flaccid abdominal wall before it do not furnish sufficient support to the diaphragm, the latter is unable to raise the ribs, but narrows the thoracic base."

FIG. 17.



Lordosis and defective posture from paralysis of the abdominal muscles.

Complete paralysis of a part of the body is easily diagnosed by reason of the complete cessation of corresponding movements. Difficulties present themselves only in conditions like infancy, deafness, unconsciousness, aphasia, psychical anomalies, etc. When a whole extremity is lost to voluntary movement, its dropping when passively raised will show it. But it should not be forgotten that many, especially children, drop the limb even when it is not paralyzed. By repeated tests, however, a conclusion may be drawn. If only certain muscles be affected, and the patient cannot be made to understand, place the limb in such a position that it can only be moved by contraction of the muscles to be examined. Paresis is harder to diagnose: the less the degree, the less certain the diagnosis. If only one side is affected, comparison with the sound side enables one to assure one's self of even a slight weakness. With children many artifices must be employed. For instance, if you are desirous of testing the motility of the extensors of the foot, stick a needle into the sole. The resulting reflex movement should not, however, be confounded with active motion.

But, providing there is no disturbance of motility, an active movement succeeds the reflex one; or it is often sufficient to merely approach the sole with a needle, allowing the child to observe your action, and it will instantly draw back its foot. It is difficult to estimate with what degree of strength plantar flexion is carried out under these conditions; yet by tickling the back of the foot in the above-mentioned manner we generally can get the child to contract his calf-muscles. It may endeavor to draw back the whole leg, but upon holding the thigh the plantar flexion follows. Paralysis of the flexors may also be recognized by the fact that when the leg is lifted vertically from the floor the foot assumes an extended position as a result of gravity.

Motor weakness or paralysis may be simple; or may be combined with decreased muscular tonicity, *flaccid paralysis*, or with increased tonicity, *spastic paralysis*.

The criteria of spastic conditions have been given above. It remains, however, to state that spastic conditions are often combined with motor weakness, even though the relations between these conditions are very changeable. Spastic paresis can affect all four extremities, but generally confines itself to the two lower limbs or to an arm or leg of one side, or it may sometimes affect one limb alone. The severer forms of this condition, in which the rigidity leads to chronic alterations in the joints, are called active or spastic contractures in contradistinction to paralytic or passive contractures. These latter cause rigidity, shortening, and atrophy of those muscles the antagonists to which are affected. This passive contracture is a complete and mechanical one; it cannot be reduced by force or pressure, is not elastic, but firmly resistant, and does not coexist with increased reflexes.

DISTURBANCES OF CO-ORDINATION.

Disturbances of co-ordination may occur synchronously with unimpaired muscular strength. A certain number of muscles of definite arrangement and grouping—all acting synergetically, yet each having its own particular function—are the conditions necessary for co-ordinated action. Whenever this legitimate interaction of movements which is governed by fixed centres is disturbed, the movements may still be executed with strength, but it is an ataxic and inharmonious action.

Incoördinated movements are not executed in a simple manner: a lavish expenditure of muscular strength is employed that not only brings into action the muscles necessary for the movement, but the impulse is carried to muscles that even inhibit those that are used, or at least do not assist them.

To examine for ataxia, have the patient placed in a recumbent position and ask him to lift a leg. If ataxia is present, it will not be raised vertically, but will vary from this position, be adducted, abducted, rotated inward or outward, and from one position be brought with a jerk into another; and, even when brought to the required position, will sway continually, especially from side to side.

When allowed to fall, it will not simply be lowered, but will be heavily dropped; not beside the other one, but across or away from it. We clearly notice that muscles not necessary for the movement demanded from the patient are brought into play. With closed eyes, this incoördination increases in intensity. When, therefore, no ataxia is noticed with open eyes, an examination with closed eyes should not be omitted.

Slight ataxia may be difficult to recognize. For this purpose more complicated tests are necessary; as, for example, to touch the knee of the recumbent leg with the heel of the other. If ataxia is present, the knee will not be touched until repeated efforts have been made. Repeat this test several times, as even normal persons may not be able to execute it at the first attempt. The patient can also be asked to describe a circle in the air with the raised leg; if ataxia is present, a very irregular figure is made with eyes shut. Comparisons with normal individuals are always advisable. With one patient dynamic tests only produced the ataxia. When I let the patient raise his leg, at the same time resisting this action, the upward swing, instead of being steady, was a swaying one. An elevated limb that continually swings, a body swaying in the erect position, or on the glutei in sitting, are all conditions of *static ataxia.* To determine ataxia of the upper extremities, let the patient touch the tip of his nose with his index-finger. If considerable ataxia exists, the finger glides past the nose, even with the eyes open, and touches the cheek or brow. The movement is also often accompanied by involuntary muscular contractions, as of the fingers or hand, etc. A slight ataxia requires an examination with closed eyes.

Ataxia has nothing to do with motor weakness. All movements may be strongly executed in spite of the incoördination, perhaps with too much strength. Close examination reveals that the strength is not rightly used. Let the leg be raised without resistance being used, and you can notice the jerks, now strong, now weak, passing from one to another of the muscular groups by which this is executed. Of course, ataxia and motor weakness may occur together, but they are separate and unassociated disturbances. Slight ataxia may even be simulated by motor weakness, inasmuch as the elevated leg, as a result of exhaustion, sways,—but, even then, not from side to side, but up and down. Further, it does not increase with closed eyes.

A very frequent, if not constant, accompanying symptom of ataxia is disturbance of the muscular sense, pressure sense, etc. The loss of these centripetal impulses are given by Leyden as the cause of ataxia. It is not impossible that centripetal excitation which does not reach the psychical centres, but merely influences muscular tonicity, may be necessary for the execution of movements, and that their absence and the hypotony or atony entailed thereby may be a cause of ataxia. (Jaccoud, Strümpell, Sherrington, Herring, etc.)

Cerebellar ataxia is distinct in itself. This refers to disturbances of equilibrium that are noticeable in walking or standing. Those affected stand with legs apart and sway from side to side in walking, like a drunken person. The name cerebellar ataxia denotes that the disturbance occurs especially in diseases of the cerebellum, which is the chief centre for co-ordination. Active movements may be influenced by another disturbance,—by

TREMOR.

This name is given to more or less rhythmical, rapid twitchings which have but a slight curve and which play in definite muscular groups (in contrast to those which pass from one group to another). As a tremor, according to the exciting cause, appears either when the body is at rest, in action, or under psychical excitement, all these conditions must be taken into account in an examination. If a tremor of the head exists, see if it still is present when the patient reclines on his back with supported head. Most tremors are recognized on inspection. When, however, they are very fine, the sense of touch must be employed to aid in locating the vibration. A hand laid on the shoulder or head, etc., will often locate the tremor. Tests with a myograph are not necessary for a clinician. Merely requiring the patient to raise or extend the extremity enables one to draw conclusions as to the influence of active movement upon the tremor. It may lessen or increase it. When a tremor is not evoked by such movements, examine by more complicated ones. Let him approach his hand to his nose, a spoon or glass to his mouth, lift a heavy weight, thread a needle, or write. A tremor of the legs accompanying active movements may be noticed in walking. In order to analyze it, let the patient while recumbent lift, flex, and extend his legs. Static and dynamic tremors can be differentiated from each other according as the tremor is found in forced positions or produced by motion. If during the examination or the conversation concerning the patient's trouble the tremor increases in intensity or only then appears, it shows the psychical influence. Other factors in its production are introspection and self-observation (auto-suggestion). This may decrease or increase it. It is important to notice the extent of each oscillation of the tremor, the rapidity with which the vibrations succeed one another, their rhythm, and regularity. In reference to the rapidity, we speak of *rapid* and *slow tremors*,—rapid when there are from eight to ten vibrations a second, slow when only from three to five. Some tremors are intermediary between these two. As to the wave amplitude, we speak of *fine* and *coarse tremors*. Generally a rapid tremor is a fine one, and we call these *vibrating tremors*.

Tests with Marey's apparatus have shown that the waves of some tremors increase and decrease with due regularity (allorrhythmical tremor), but this knowledge has not been of diagnostic importance.

A tremor which depends upon increased tendon-reflexes is called *spastic* if it occurs during some motion; as, for instance, ankle clonus, which depends upon active raising of the toes.

A special form of tremor is the *fibrillary*. A twitching of a single muscular bundle, or a wave which attacks rapidly one fasciculus of a muscle after another, so that the whole muscle glimmers and ripples, is noticed in this type. In emaciated persons cold or denudation will produce the same phenomenon. Bodily over-exertion may also evoke this tremor. Severer forms are noticed only under pathological conditions; in such cases extraneous influences, as cold, are not necessary for their production, though they may be increased thereby. Fibrillary twitching may sometimes be observed only after electrical stimulation, the resultant muscular contraction being succeeded by an undulation of the muscle which lasts for some time. It should be remembered that healthy persons, through exertion, cold, strong emotion, in convalescence, and from excesses of every kind, may also tremble. In a tremor from the effects of cold it is easily noticed how volition may influence the tremor. Chattering and rattling of the teeth is the more prominent and more intense the more one gives way to his feelings and the less one endeavors to repress the phenomenon. In some forms of pathological tremors, especially hysterical ones, something similar may be noticed.

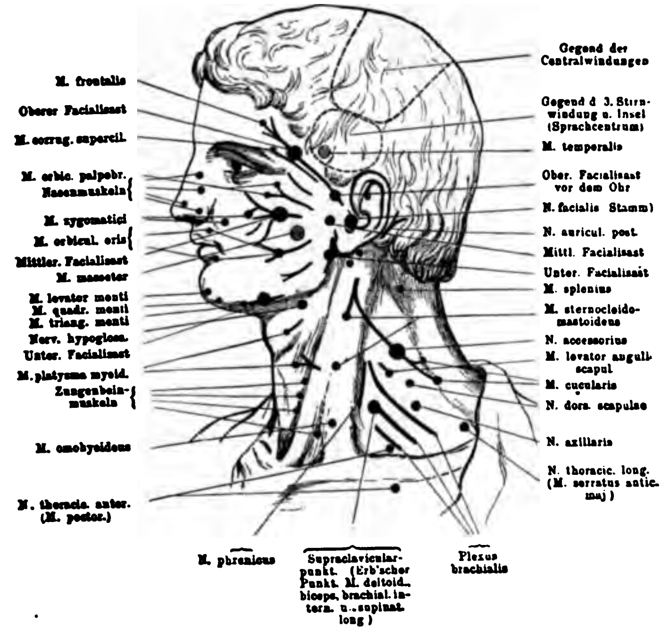
Smoking and drinking may evoke a temporary or persistent tremor. It is almost always a symptom in chronic alcoholism. For descriptions of tremors caused by other poisons and accompanying certain diseases, see the special part of this book. (The chapters on Multiple Sclerosis, Paralysis Agitans, and Hysteria are particularly to be noticed.) There is also an *inherited* tremor that is transmitted for generations without any other symptom accompanying it. Its attributes are so variable as to leave nothing characteristic to be said about it.

Having informed ourselves as to the behavior of the active movements, we next desire to determine whether we are dealing with a simple, degenerative, or functional paralysis. For this purpose an electrical examination offers us the most aid.

ELECTRICAL EXAMINATION.

In electrical examinations the following apparatus is necessary: (1) an induction apparatus of one or two elements; (2) a galvanic battery sufficient to furnish a current of at least thirty milliamperes, and equipped with an absolute galvanometer and a commutator; (3) a pair of good (undefective) induction cords; (4) a number of electrodes of different sizes (the largest with an area of from fifty to seventy square centimetres, the smallest with a diameter of from one to three centimetres, the normal electrode being ten square centimetres). A wire brush or pencil is necessary for some examinations. (Fig. 18.)

FIG. 18. (After Erb.)

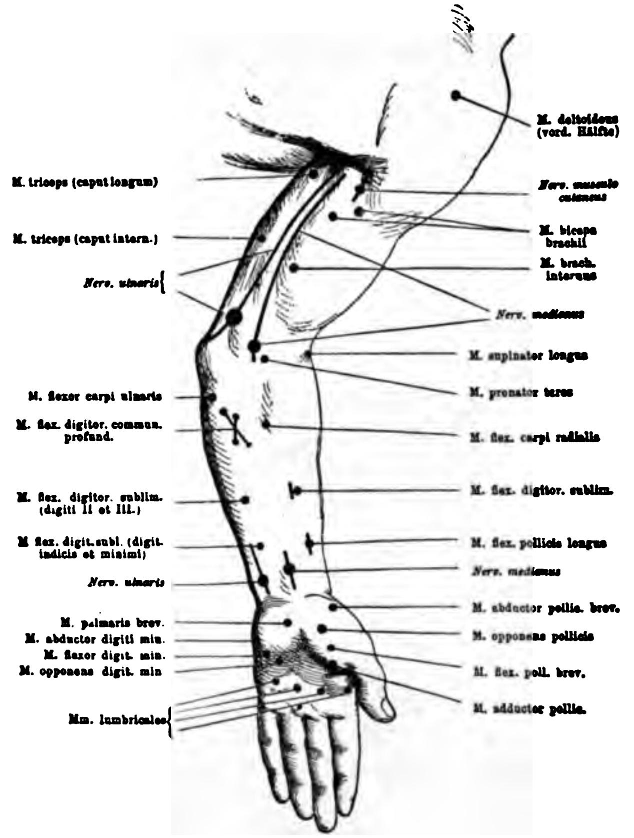


A knowledge of the motor points is the first requisite in an electrical examination. By motor points we understand the places where the nerves and muscles can be most easily stimulated. The figures show the position of these points.

The covering of the electrode should be thoroughly wetted. A single dipping in warm water is not sufficient, it must be moistened through and through. Especially when unused for a considerable time it is so dry that it has an almost insurmountable resistance.

The large (fifty to seventy square centimetres), indifferent electrode should be firmly applied to the lower part of the sternum or to the neck. It can be fastened there, or the patient can hold it, but it must not be removed during the examination.

FIG. 19. (After Erb.)



The electrode that is to be applied (it should be small, about ten square centimetres, and have an interrupter) is grasped in the right hand with the thumb upon the interrupter, and then, the current being open, placed firmly, with its whole surface upon the place desired, the current being closed by lifting the finger and instantly opened again.

If a contraction does not ensue, a slight displacement of the electrode only may be necessary to secure an effect. If this is not the case, the strength of the current is to be gradually increased.

Commence the examination with a (secondary) faradic current and endeavor to determine the minimal current strength which is required for stimulation.

FIG. 20. (After Erb.)

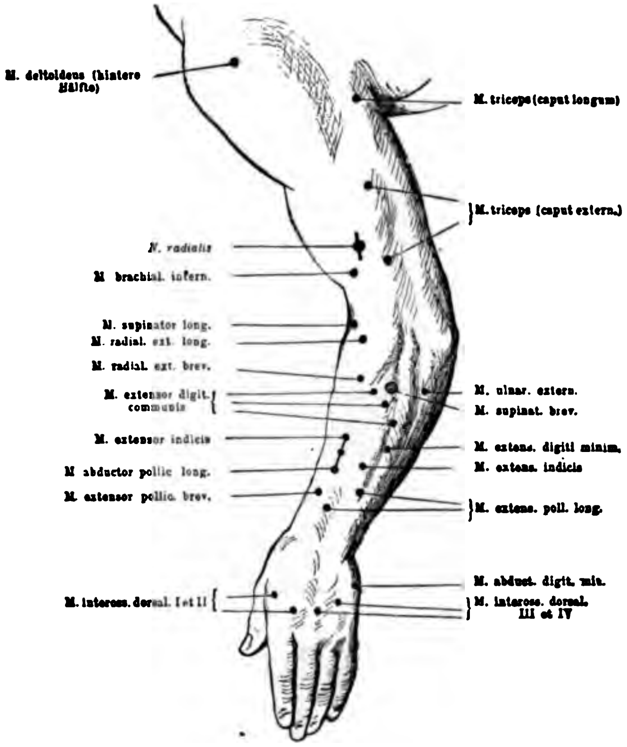


FIG. 21. (After Erb.)

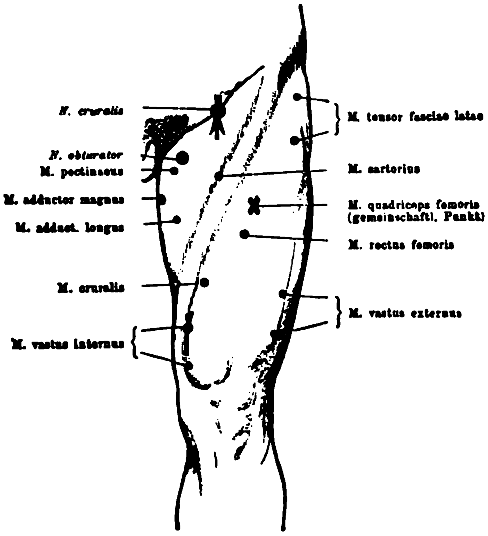


FIG. 22. (After Erb.)

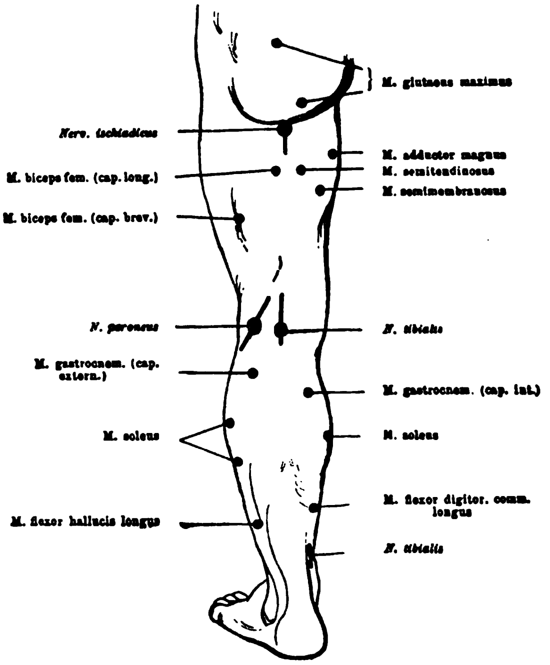
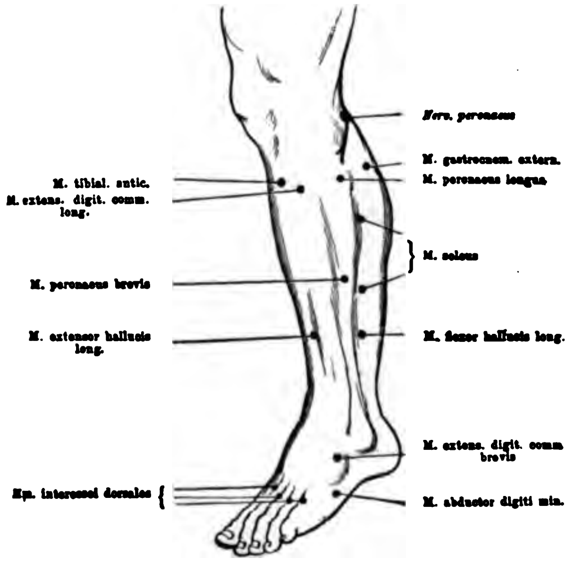

M. glutaeus maximus
Nerv. ischiadicus
M. adductor magnus
M. biceps fem. (cap. long.)
M. semitendinosus
M. semimembranosus
M. biceps fem. (cap. brev.)
N. peroneus
N. tibialis
M. gastrocnem. (cap. extern.)
M. gastrocnem. (cap. int.)
M. soleus
M. soleus
M. flexor digitor. comm. longus
M. flexor hallucis longus
N. tibialis

FIG. 23. (After Erb.)



Let us imagine that the arm is to be examined.

First, we should determine the minimum stimulability of the nerves in the following order:

1. Erb's point.
2. Musculocutaneous nerve.
3. Median nerve.
    - Upper point.
    - Lower point.
4. Ulnar nerve.
    - Upper point.
    - Lower point.
5. Radial nerve.

As soon as a twitching is noticeable, the required current strength has been reached which we were desirous to determine; for example:

Erb's point, one hundred and twenty millimetres.

Median point, one hundred and twenty-five millimetres.

Next comes the direct muscular excitation, in which a somewhat larger electrode is required. The diameter of the applied electrode must always be noted, as the effect of the stimulation depends upon the density of the current as well as upon its strength (that is, the density of the current is proportional to its strength and inversely proportional to the diameter of the electrode).

The extremity is always to be placed in that position in which the effect of the stimulation can most readily be perceived; for example, in exciting the extensors, flex the hand; in excitation of the extensor communis digitorum, flex the basal phalanges of the fingers, etc. The patient should also guard against active contraction, because he might thus lessen or conceal the effect of the stimulation. It is very difficult to use electricity upon small children, as they cannot hold their limbs quiet, and it is difficult to distinguish contractions produced by the electricity from volitional and reflex movements. In such cases the extremities must be firmly held by a third party. If there is any doubt as to whether the muscular contractions are produced by electrical stimulation, the faradic current is to be kept closed for some time; the muscle then becomes tetanically contracted.

Undue development of the fatty tissue renders examination of children difficult. In the new-born, according to the observations of C. and A. Westphal, the nerves and muscles react only with a strong current, and then but slowly. This is a result of the high resistance offered by the skin, and also of the incomplete development of the peripheral nerves (especially as regards medullation) and of the muscles. Not until the fifth year do the results approach those of adults.

Galvanic Examination.—Apply the cathode to the different points, as in faradic examination. Provided there is any doubt as to the polarity, the cathode can be recognized by immersing the poles in water, when the introduction of a strong galvanic current will cause a lively production of gas at the cathode pole.

Introduce a mild current and gradually increase it until closure produces a contraction, noticing especially whether all the muscles supplied by the nerve take part in the contraction. At the moment the closing is made introduce the galvanometer into the circuit and observe the needle-deflection. The number of elements used need not be noticed.

The result of such an examination will be something like the following:

Erb's point, *CaCC* (cathode closure contraction), 2.0 M.A. (milliamperes).

Median nerve, *CaCC*, 0.8 M.A., etc., etc.

The contractions are normally *short* and *lightning-like.*

Next follows the direct galvanic muscular stimulation, in which it is necessary to observe whether the *CaCC* is stronger than the *AnCC*, the rotations on the commutator being carried out at the same time. Of less importance is the behavior of the muscles in regard to the opening contractions. The manner and the character of the contractions are also to be noticed; normally lightning-like and rapid, they may deviate widely from this in disease.

It is easily seen that through the influence of galvanic currents the resistance of the skin decreases. The longer manipulated, the longer the current is closed, the stronger the contractions become, and one must lessen the current strength by removing elements from the circuit, or suddenly insert some resistance to detect the minimal contractions. Also, after calculating the minimal contraction by bringing the resistance made by the galvanometer suddenly into the circuit, the current strength is lessened.

The *changes of electrical irritability* through disease consist in,—

(1) *Quantitative decrease or increase.*

(2) *Quantitative and qualitative* or *modal and serial* changes.

Simple decrease of irritability is recognized by the fact that a stronger current than normal is necessary to produce contraction, or a current that is ample to produce contractions in sound nerves and muscles fails to have an effect. This is not difficult to observe, providing the changes in the muscles occur only on one side and the other can be used as comparison. But even in this case one may be deceived by the differences in resistance of the skin which may occur between the two sides. It is therefore necessary to determine, first, the resistance of the skin of the two sides. Only when the decrease is considerable, and when noticeable changes of the skin (scars, edema, cyanosis, etc.), are absent, can these comparisons be of any value. In changes on both sides, a third person must be used for purposes of comparison; but this is a very inexact method and very uncertain. Stintzing has published results of examinations of the irritability of the nerves and muscles of normal individuals. In order that these, as much as they refer to the faradic current, may be of value, one must use the same apparatus. Stintzing found that the different degrees of excitability of a nerve (and muscle) did not vary much, the maximal difference in different persons for the same nerve being about twenty-one millimetres. It should, however, be understood that the Stintzing electrode of three square centimetres is to be used.

We take the following from Stintzing's tables for the

Faradic Irritability of the Nerves.

NERVE.	LOWEST.	AVERAGE.	HIGHEST.	MAXIMAL DIFFERENCE OF THE TWO SIDES OF THE BODY.
Facial	145 mm.	132—110	102	10
Accessory	. . . .	145—130	125	10
Median (in sulc. bic. int) . .	141 mm.	135—110	100	12
Ulnaris I. (Above olecranon)	145 mm.	140—120	110	6
Ulnaris II. (Between olecranon and int. condyle) . . .	. . . .	130—107	. .	11
Radial	125 mm.	120— 90	. .	16
Crural	. . . .	120—103	. .	8
Peroneal	138 mm.	127—103	95	13
Tibial	125 mm.	127— 95	93	10

In practice every one becomes gradually accustomed to his own apparatus, and through experience is able to tell what strength of current is necessary for normal nerves and muscles. It is advisable, however, only to lay stress upon large differences and then only with corrections according to the resistance of the skin. The faradimeter to test the absolute current strength has not been found of any value by me. The irritability of the muscles varies more than that of the nerves.

Quantitative decrease of faradic stimulability is recognized by the fact that less current strength is necessary than in normal individuals to produce the first noticeable contraction.

In testing the galvanic irritability the absolute galvanometer enables us to take exact measurements and to be able to observe extreme quantitative changes. Before we, however, enter into a discussion of this, it is of advantage to learn the laws of contractility as much as they concern living tissue. The conceptions of physiology concerning ascending and descending currents must be ignored. We use only one electrode for stimulation; the other rests upon the sternum. It follows that the nerve and muscle react only to alterations of the current, especially in the opening and closing of the current. In using a weak current, they react only in the moment of the closing through the negative pole (*CaCC*). With increase of the current, the positive pole in closing as well as opening evokes a contraction (*AnCC* and *AnOC*, the first generally before the latter). With further increase of current there takes place at the moment of the closing, in using the cathode, a tetanus, cathode closing tetanus (*CaCT*). The muscles remain in a tetanic contraction as long as the current is closed. Finally, there follows a *CaOC* and an *AnCT*, but only from such a strong current that they can be ignored clinically. An *AnOT* cannot be produced in normal individuals. These phenomena are easily demonstrated in normal individuals; select the ulnar nerve, place the electrode (open) upon the nerve, without displacing it, commence with a weak current, using the cathode, observe the minimal *CaCC*, change the current, and observe all the other phases of the law of contraction.

To evoke the *CaOC*, wait until the tetanus produced by the closing is passed, or by gradual increase of the current prevent it altogether.

In direct stimulation of muscles the law of contraction remains unchanged, only the muscle reacts less to stimulation with an open current; it occasionally happens, too, that the *AnCC* may produce in normal muscle a contraction just as strong as or even stronger than the *CaCC*.

Quantitative decrease of galvanic irritability is recognized from the fact that the first *CaCC* only appears with a stronger current,—*i.e.*, more milliamperage than normally, and that the higher phases (*CaCT*, etc.) cannot be produced. Erb proved that on most of the superficial nerves the first *CaCC* appeared with 0.5–2.4 M.A.

Galvanic Irritability (*CaCC*). (*After Stintzing.*)

NERVE.	MINIMAL VALUE.	AVERAGE VALUE.	MAXIMAL VALUE.	MAXIMAL DIFFERENCE BETWEEN THE TWO SIDES.
	Milliamperes			
Facial	0.8	1.0 —2.5	2.8	1.3
Accessory	. .	0.01—0.44	0.6	0.15
Median	0.27	0.3 —1.5	2.0	0.6
Ulnar I.	. .	0.2 —0.9	1.3	0.6
Ulnar II.	. .	0.6 —2.6	. .	0.7
Radial	0.7	0.9 —2.7	3.0	1.1
Crural	0.3	0.4 —1.7	2.6	0.6
Peroneal	. .	0.2 —2.0	2.7	0.5
Tibialis post.	. .	0.4 —2.5	. .	1.1

Stintzing's tables, provided one uses the same procedures, may serve as a criterion for the irritability, but it is advisable only to lay stress upon large alterations and regard only such as pathological. The variability in the thickness of the skin and the fact that the nerves lie more superficial in some than in others, produce marked differences. For example, a difference of 1 M.A. (*CaCC*) between the facial of the sound side and that of the diseased side is not conclusive of anything. It is also necessary to satisfy one's self whether other circumstances may not deceive and cause alterations.

Example of Quantitative Decrease of Irritability.—Inactivity-Atrophy of Right Arm.—Large Electrode of Seventy Square Centimetres upon the Sternum.—Electrode of Three Square Centimetres for Nerve and Ten Square Centimetres for Muscular Excitation:

	RIGHT ARM.		LEFT ARM.	
Erb's point	90 mm.	5.0 M A. (CaCC)	130 mm.	3.0 M.A. (CaCC).
Median	98 mm.	6.0 M.A. (CaCC)	120 mm.	1.5 M.A. (CaCC).
Ulnar	90 mm.	4.0 M.A. (CaCC)	140 mm.	1.0 M.A. (CaCC).
Radial	80 mm.	6.5 M.A. (CaCC)	110 mm.	2.5 M.A. (CaCC).
Deltoid	85 mm.	14.0 M.A. (CaCC>ACC).	100 mm.	8.0 M.A. (CaCC).
Biceps	100 mm.	7.5 M.A. (CaCC>ACC).	130 mm.	3.0 M.A. (CaCC).
Sup. long.	90 mm.	8.0 M.A. (CaCC>ACC).	120 mm.	5.0 M.A. (CaCC).
Ext. carpi rad.	100 mm.	7.0 M.A. (CaCC>ACC).	110 mm.	4.5 M.A. (CaCC).

Simple decrease of excitability occurs in inactivity-atrophy, hysterical muscular atrophy, primary muscular affections, as progressive muscular dystrophy, myositic atrophy, atrophy following muscular compression as a result of trauma, tumors, etc., in muscular atrophy accompanying joint-disease, and, lastly, in slight peripheral neuritis.

Quantitative increase of the electrical irritability occurs seldom. In this condition the first noticeable contraction occurs with a current strength that is less than normal, as, for example, with 0.05—0.1 M.A.; and with a normal current, the intensity of the contraction is increased. It is distinctly noticeable only with the galvanic current, in the use of which the higher phases of the law of contraction (*CaCT*, *CaOC*) appear already with a weak current, and reactions like the anode opening tetanus are obtained.

This increase only appears prominently in one disease, tetany, and we will speak of it there.

The *reaction of degeneration* (*DeR*) is, for diagnostic purposes, the most important form of the alterations in excitability.

A complete degenerative reaction presents the following phenomena:

1. Excitability of the nerves for the faradic current is lost.
2. Excitability of the muscles for the faradic current is lost.
3. Excitability of the nerves for the galvanic current is lost.

4. The excitability of the muscles for the galvanic current is (*a*) increased; (*b*) modified so that the contractions become slow, and the *AnCC* is stronger than the *CaCC* (*AnCC*>*CaCC*). (The *CaOC* also increases more than the *AnOC*, and can become equal or stronger than it,—a factor which can be generally ignored.) Of all of these, the characteristic phenomenon of the *DeR* is the increase of galvanic irritability in the first stage; it gradually decreases so that finally (often only after years) a weak and slow *AnCC* with strong current is the only residuum of the disturbance. This condition is often overlooked by the beginner. If the contraction does not appear, use a very strong current, rotate the commutator, or stroke the skin slowly with the electrode, and watch the muscle, not the limb, for the locomotor effect of this contraction often fails or is very slight, often only a slight muscular wave being produced.

The preponderance of the *AnCC* over the *CaCC* should not be regarded as being always present, as the relations are often reversed. We can speak of *DeR* also when the *CaCC* is equal to or exceeds the *AnCC*; *the most important part is the sluggish contraction.* There exists, also, a *partial DeR* in which the nerve irritability is but slightly lowered, the faradic irritability of the muscle being also decreased or lost, while, in direct galvanic stimulation, the slow contraction and the reversal of the formula appear as prominent symptoms.

There exist all imaginable degrees between the partial and complete *DeR*. Stintzing, for example, differentiates thirteen varieties of *DeR*. Among others, the muscular contractions occurring from nerve stimulation may become sluggish (*partial DeR*, with indirect sluggishness of contractility). A sluggishness of the contractions in faradic stimulation of the nerves and muscles has also been observed (*Remak*), which, however, may be the result of cold, and is not of much interest.

I have often found sluggish contractions, in polyclinic examinations, which could not later be demonstrated in the lecture-room to my audience because the disturbance caused by the cold disappeared in the mean time in the warm room.

It is easily understood that the less complete the *DeR* is, the more difficult is the diagnosis; and it is just those cases of partial *DeR* which are easily overlooked. Observe especially sluggish contractions with direct galvanic stimulation, and endeavor to recognize them, making comparisons with healthy muscles.

DeR is the most certain and constant sign of degenerative changes in the motor nerves and muscles. Experimentally, it can be produced by section or laceration of a nerve. Changes of degeneration in nerves and muscles run parallel with these alterations of electrical excitability, and, like them, are only completely developed in seven to ten days.

The *DeR* is, therefore, not to be expected for a week. We find it in all severe diseases of the peripheral nerves (motor or mixed), as well as in diseases of the anterior horns and roots; everywhere where the trophic centres of the muscles or where the conducting tracts between them and the muscles (anterior muscle, peripheral nerve) are attacked by a deeply invading disease-process.[1]

These are

I. Diseases of the *anterior horns.*
   1. Poliomyelitis anterior (acute).
   2. Poliomyelitis anterior (subacute and chronic).
   3. Amyotrophic lateral sclerosis.
   4. Spinal form of progressive muscular atrophy.
   5. Spinal gliosis.
   6. Diffuse cervical myelitis, lumbosacral myelitis.

I*a.* Diseases of the *bulbar nerve nuclei* corresponding to the anterior horns (progressive bulbar paralysis, acute inferior polioencephalitis).

II. Diseases of the *anterior roots.*
   1. Compression from tumors and tumor-like thickened membranes; for example, in lues, hypertrophic cervical pachymeningitis.
   2. Compression in the intervertebral foramina in vertebral diseases (caries, tumors, fracture, luxation).

III. Severe diseases of the *peripheral nerves.*
   1. Traumatic (pressure from tumors, bruises, incision, etc.).
   2. Rheumatic (for example, facial paralysis).
   3. Toxic and infectious.
      a. Lead paralysis.
      b. Alcoholic paralysis.
      c. Arsenical paralysis.
      d. Infectious forms of multiple neuritis, etc.

Wherever an incomplete *DeR* is found, the possibility of regeneration and restoration again of a part must be borne in mind. It is further important to know that, after restoration of the function, electrical stimulation often is impossible for a long time, while the will may produce a contraction of the muscle. Other rarer forms of alterations in contractility, as the myotonic and myasthenic reaction, will be spoken of in the Special Part.

Some points for diagnosis are obtained by the

---

[1] Only in a few cases was a partial *DeR* found also in primary muscular diseases (dystrophy, trichinosis).

EXAMINATION OF THE MECHANICAL IRRITABILITY OF THE MUSCLES AND NERVES.

Percuss the muscle of a sound person, and either no contraction or merely weak, short contractions are noticeable. Occasionally one is also able, especially on the biceps, to produce "idiomuscular contractions" by strong stimulation. In emaciated persons, especially consumptives, this mechanical excitability is considerably increased : contractions may be produced at any point. If, for example, you stroke with the point of the percussion hammer the pectoralis major, the muscular bundles touched contract one after another as noticeably as if one grasped the strings of a harp (harp phenomenon). We find increased mechanical irritability in those nervous diseases which present a general increase of excitability (neurasthenia, traumatic neuroses, etc.). Chronic alcoholism and rheumatism may produce the same phenomenon.

Pathologically, the idiomuscular contractions may follow the slightest stimulation ; the intumescence caused by the slight blow may also last several seconds. The meaning of this phenomenon is not understood.

In cases of degenerative atrophy, the contractions produced by mechanical stimulation, where galvanic hyperexcitability is present, are noticeably delayed (mechanical reaction of degeneration). The peripheral nerves may also be excited by mechanical stimulation. Percuss the ulnar nerve or roll it upon its bony floor with the finger, and, in many persons, a slight contraction of the corresponding muscles is produced. Under pathological conditions this contractility can be considerably increased, the most constantly and markedly in tetanus.

There exists also an increase of the mechanical irritability of sensory nerves, which, however, has no known diagnostic importance.

EXAMINATION OF THE GAIT—DISTURBANCES OF WALKING.

Examinations of the simple active movements in the reclining position are often not sufficient, and must be supplemented by examination of the complicated functions, gait and sway. Even though disturbances of gait may have other causes and have no reference to the condition of the motility, we will, nevertheless, enumerate here the most important points concerning this function.

First, we should remember that there are normally many physiological variations in gait. We do not all walk alike. One rocks the hips from side to side, another lifts the pelvis up and down (women with large abdomen), etc. All these peculiarities must be taken into account.

The Simple Paretic Gait.—The disorder of gait produced by simple muscular weakness is shown by slowing of the walk and shortening of the strides. The active movements of the legs are also less yielding, and the muscular weakness can produce an exaggerated but purely mechanical flexion of the knee-joint. In marked grades of this condition the patient drags himself wearily along and is compelled to use his upper arms for support. By the use of a support to these (crutches), walking, even with complete paralysis of the legs, is possible. We then can say that the patient walks with his arms. Lately I saw in the Tyrol a man climbing a high mountain in this manner.

The Partial Paretic Gait.—This form is considerably modified when only single muscles or muscular groups are paralyzed (see the chapter on muscular functions); a very typical and frequent form is the double peroneal paralysis. The foot of the swinging leg falls with the toes pointing downward—a result of gravity. As the leg is thereby lengthened, the patient, to overcome the effect of this, must flex the extremity at the hip-joint and knee-joint. Setting down the foot causes two sounds. The gait has been compared to that of a horse.

The Spastic Paretic Gait.—If muscular stiffness is combined with the muscular weakness, a new difficulty presents itself. The stiffness induces slowing of motion and a decrease of each excursion. The leg is propelled forward to a certain degree like that of a goose, or like a cord held taut from behind. Especially characteristic is the sliding of the toes along the floor, which produces a creaking noise. This is caused by the fact that the contracture of the calf muscles is overcome with difficulty by the extensors of the foot and toes. Deficient movements of the joints of the leg are generally compensated by an elevation of the pelvis on the side of the swinging leg. In the highest grades of this condition the toes are, to a certain extent, bound fast to the floor, and remain constantly in contact with it, the patient pushing himself forward in small strides. If the adductor contracture of the thigh is in the ascendancy, as is generally the case, the knees rub against each other and the thighs become crossed in walking.

The Ataxic Gait.—In pure (spinal) ataxia the disorder of gait is typical, and especially characterized by the excessive excursion of each motion.

The swinging leg is flexed and rotated outward at the hip-joint more than normally; this movement is brisk and irregular, the toes being strongly raised at the same time and the leg thrown down with such force that the heel strikes the floor with a stamp. The gait is uncertain, the legs are spread apart, and the walker must constantly eye the floor and is in danger of falling as soon as he looks away.

A modification of this gait is the *cerebellar-ataxic*. We differentiate two forms which at times combine,—(1) one depending upon (vertigo and) incoördination, which resembles greatly the gait of a drunkard, inasmuch as the individual reels, sways from side to side, and staggers; (2) one depending upon ataxia. The patient walks with legs apart and stamping, but without much reeling or irregularity. He stands with legs apart, and one sees a continual balancing, a momentary contraction of the extensors in constant repetition. A sharp distinction between this gait and the spinal ataxic is possible only when the modification described first is combined with it.

The Disturbance of Gait caused by Tremor.—The tremor can attack the legs and increase so much in walking that every simple muscular action is replaced by a tremor. Indications thereof are already observed in a spastic gait, inasmuch as the placing of the foot or toes on the floor produces a clonus which shakes the whole body. Under other conditions (multiple sclerosis) the whole extremity waddles, or a complete "shaking-palsy" takes place (hysteria).

If the tremor attacks especially the body and head and consists of large oscillations (as in multiple sclerosis), a noticeable unsteadiness in walking is produced; the patient may fall over after taking a few steps. Not rarely we find a combination of the different forms, especially the spastic-paretic with the ataxic.

Very variable are the disorders of gait caused *by pain;* it is not possible to describe them. If pain occurs upon contact of the toes with the floor, the patient walks on the heel, and *vice versa.* When the whole sole is hypersensitive, he either avoids using it altogether or, walking very carefully and slowly, seeks to step as lightly as possible, and makes painful grimaces at every step. The gait then alters itself according to the location of the pain.

Imaginary inability to walk may also influence the gait. The inability to walk with unimpaired movement of the legs in the reclining position is called abasia.

Further particulars as to the disturbances of gait in chorea, paralysis agitans, etc., are given in the special part.

EXAMINATION OF THE SENSIBILITY.

The simplest methods of examination are the best. All esthesiometers are better left alone. The exact measures used by physiologists are impracticable at the bedside. We use for examinations of sensation a brush, a needle, and a test-tube of hot and one of cold water. It is necessary to know that normal persons, by giving sufficient attention to it, are able to experience all over the body contact with a soft substance, as a brush or the finger-tip. Only where scars and hypertrophies are found, as the ball of the great toe of many individuals, are these weak sensory stimulations not felt. Pressure with a hard substance (as the handle of a brush) is also differentiated from touch with a soft one in all places except where the epidermis is excessively thickened.

In the examination let the patient close his eyes, and then lightly touch his skin at different places; he is to denote the moment of touch by "now." In order to know whether those who do not give the word have any disorder or are merely inattentive, some part which certainly is not anesthetic is touched occasionally. This prevents being deceived.

A needle-prick produces everywhere and with every one a *feeling of pain.* Its intensity, of course, is variable. All parts of the body do not have the same degree of sensibility to pain; it is not so marked on the scalp, tongue, and dorsal surface of the lower arm. In early childhood the sensibility for painful stimulation is only slightly developed.

Whenever pain from a needle-prick is less acute by reason of disease, it occasionally is possible to produce pain by drawing the needle in a long sweep over the skin. Here, however, we have not a single stimulation, but a summation of stimulations.[1] The same may be said of the faradic brush. Use a current strength which will evoke pain on your own skin or on the healthy parts of the patient, and compare with the sensitiveness of the affected parts. By sudden closing of a strong galvanic current, with the use of the brush as cathode, one can produce a very intense pain which is absent only in marked disorders. Electro-cutaneous examination of the sensibility by measuring the current strength which produces noticeable prickling of the skin has no advantages over simpler methods. When asking to distinguish the head from the point of a pin, not one but more attributes of sensibility are tested,—touch, pressure, pain, as well as the ability to recognize impressions according to their intensity (sense of size), as the point stimulates a smaller area of the skin than the head. If you use an ordinary pin, you must prick pretty severely to produce differences in all regions. Even in normal persons, especially on the back, the examination may, however, not be accurate.

We do not possess exact methods for the determination of pain. The *algesimeter* is not practicable. Lately, *Moczutkowsky* has recommended a similar instrument and with it made observations on the normal skin as to the degree of pain in different regions. He finds the least sensibility in the pelvic and gluteal region, and that the sensibility increases towards the head and fingers and towards the toes. The frontal region is the most sensitive. He has given exact figures as to the sensibility, but further observation is necessary to confirm his data.

---

[1] The rhythmical repeated weak stimulation that produces periodical pain in certain diseases is also caused by a summation (*Naunyn*).

Sticker's attempts to present the sensibility objectively have not as yet succeeded.

To test the *temperature sense* touch the skin with test-tubes filled with hot and cold water. It is recommended not to use the highest degree of temperature, as this produces pain. Remember that warmth becomes absorbed but slowly by the skin, and therefore allow the test-tube to remain in contact some seconds. If the temperature of the skin is much decreased, as in frost, the temperature sense is lessened.

Goldscheider's more exact method of testing the temperature is of little clinical value on account of its awkwardness and the demands it makes on the patient. The data upon which it is based are, however, so important that we will discuss them here. The skin has special nerves for pressure, cold, warmth, etc. (Blix, Goldscheider). These end in the skin in distinct and separated points. Upon the pressure-points the sensitiveness for mechanical stimulation is especially well developed. Stimulation of a certain intensity produces upon these, as well as upon the skin lying between them, pain. Special pain-points are not found. Prickings with a needle are more painful at some spots than at others, but this is probably due to especially exposed nerve-endings (Goldscheider) and cannot be regarded as evidence of the existence of special nerves for pain. We also do not know what nerve-endings are intended for the different forms of sensibility.

Cold and warm points are not equally distributed over the body. The sensibility for temperature is unequal at different places on the skin.

The differences are, however, according to Goldscheider, constant, and he has divided the body-surface into twelve areas of different degrees of sensibility to warmth, and eight of cold, each presenting topographical differences. His method of examination is to compare the temperature sense of a part of the skin in question with the same area of a normal one, and, when differences exist, endeavor to find the degree which corresponds to the skin-area under examination. He uses a solid metal cylinder of about one square centimetre in area, which has a handle of ebony. The cylinder by douching in cold water or heating in the flame is brought to the temperature desired.

To examine for the *sense of place*, confine yourself to asking the patient to locate the places of stimulation. Even in normal persons, exact answers cannot be expected. A difference of one centimetre on the hands, or from two to four on the arms and legs (according to Ziehen and Levy, six or seven on the upper arm and thigh) is not pathological. The exactness of localization depends also upon the intensity and length of the stimulation. Localization is most exact on the face.

Another method of testing this sense is by examining the ability of detecting stimulation produced by two simultaneous applications. This is very variable: two points only one millimetre apart can be detected on the tongue, while on the back they must be separated sixty-five millimetres. This method is, however, very inexact.

It is necessary to examine the relations of sensibility in the deeper parts of the body (joints, fasciæ, muscles). We therefore examine the *feeling for passive movement and the sense of position,—i.e.*, we endeavor to tell whether the patient feels our moving his limbs slightly and whether he can locate the position of the limb thus moved. It is necessary that the passive movements executed be very limited in extent; for instance, the great toe, which is in a position of flexion, should be slightly extended, at the same time avoiding any pressure upon the skin. If during extension the skin on the flexed side is pressed, or *vice versa*, the patient can form conclusions as to the direction of the movement; therefore we should grasp the end phalanx between thumb and index-finger and exercise, above and below, equal pressure when executing a movement. In the same way examine the other joints. The patient also should not support the passive by active movements, which he is inclined to do.

Goldscheider has made the following observations concerning the sensitiveness of movement of the different joints as observed with a certain curve of movement.

Second phalangeal joint of the index-finger	1.0° — 2.0°
First phalangeal joint of the index-finger	0.7° — 1.0°
Wrist	0.3° — 0.4°
Shoulder	0.2° — 0.4°
Hip	0.5° — 0.8°
Knee	0.5° — 0.7°
Ankle	1.0° — 1.3°
Metatarso-phalangeal joint of the great toe	2.0° — —

He has constructed an apparatus for the testing of this sensibility. We can, however, dispense with it at the sick-bed, as with normal individuals these feelings are so sensitive that a movement that can be seen or felt by the examiner will produce an impression upon the patient.

The position in which we place the extremity, eyes being closed, is to be noticed by the patient either in writing or by denoting the direction with the hand. For example, we lift the leg, rotate it inward, and ask the patient to indicate with his index-finger the position in space of the large toe. If the disturbance is unilateral, he can be asked to indicate with the sound limb the position of the other.

Examination of the *sense of force and weight*[1]—the ability to tell the weight of elevated bodies—can generally be disregarded. The method of the examination is the following: Tie a cloth around the limb so that the sling hanging from it will hold weights. (For the lower limb a stocking with a pocket sewed to it has been recommended.) Endeavor to discover what differences in weight will be detected. Tests on normal persons vary so much that conclusions can only be drawn with difficulty. The sense of weight is more acute on the upper than on the lower limb. Upon the upper, differences of one-tenth (ninety from one hundred grams), can be distinguished. On the legs, Hitzig distinguished zero from one hundred but not from ninety grams, two hundred from two hundred and fifty, and differences of weight from one hundred to one thousand grams correctly. According to Chavet we can detect one gram differences on the upper extremity, but only thirty to forty on the lower. As the results vary so much in healthy persons, only gross differences should be considered pathological. Hitzig uses balls of equal size but of variable weight, being filled with varying amounts of lead (Kinesiesthesiometer).

An examination of the *stereognostic* sense may be necessary. To do it methodically, use geometrical bodies, preferably of wood, from three to six centimetres in diameter,—balls, half-spheres, octahedrons, etc. The healthy individual recognizes them as soon as they are placed in his hand, even with his eyes shut. We may also use small, easily recognizable substances, as money, watch-key, buttons, etc. It is not, however, a simple sense, but a combination of sensibilities, especially pressure, passive movement, and position of limb.

The stereogenetic sense may be absent, therefore, when the pain and temperature senses are present. In contradistinction, I found it often lessened with disorder of the sense of position alone.

In all such examinations it should not be forgotten that the patient must be attentive; if he is not intelligent or is inattentive, or if some disturbance of the sensorium is present, dexterity and patience are necessary to form any reliable opinion as to the condition of the sensibility. To be certain that the patient's attention is not distracted, occasionally stimulate the sound skin and request him to point out with his finger the location of each stimulation. Even with small children this is to be recommended. It interests them more to point with their finger than simply to say "now" every time. Do not continue the examination too long and tire the patient. On the other hand, do not lay too much stress upon first tests. Even with normal persons the first needle-prick, especially on the leg, may not produce any pain, while all succeeding ones do. There are also certain anomalies of sensibility which may equalize themselves as a result of the examination,—*e.g.*, double-feeling, delayed sensation, and disturbances of the sense of position.

---

[1] The name *muscular sense* had best be dropped. According to the definition of Goldscheider, this includes:

1. Sensation of passive movement.
2. Sensation of active movement.
3. Sensation of heaviness and resistance (strength).
4. Sensation of position.

It often happens that the paresthesia is so distracting that the patient cannot distinguish the subjective from the objective stimulation. The fact that all stimulation is noticed is no indication of normal sensibility. Always try to see whether stimulation is as intense as in regions where no disturbance is expected. When only one side is affected, always make comparisons with the other.

In certain brain-diseases which cause unilateral disturbance of sensibility the following procedure is advantageous: Stimulate simultaneously two symmetrical points. The patient will always detect it only on the sound side; whereas in single tests he may detect every stimulation of the affected side. This manner of examination we would call the *method of double stimulation*.

DISORDERS OF SENSATION

are subjective and objective. Under the subjective are included pain and paresthesia. It is not necessary to dilate upon the nature of pain. Only certain points which have a bearing upon the diagnosis will be noticed. The exclamation, "I suffer pain," should not satisfy the physician. It is important to learn its character, extent, onset, duration, accompanying phenomena, and the influence of the pain upon the general condition.

In accounts of the intensity of pain, the subjective element is naturally of chief moment. The same excitation may cause slight pain in one, severe in another; and also pain may occur in the centres without the end organs or the conducting tracts being stimulated. Severe pain generally produces certain accompanying symptoms in the motor, vasomotor, and secretory organs which are not dependent upon the will (contraction of muscles, flushing or paling of the skin, watering of the eyes, tachycardia or bradycardia, etc.); even delirium may be produced. It is advisable to become certain as to its extent, whether it follows certain tracts, spreads in circles or bands, or is confined to one spot, etc. *Topoalgia* (*Blocq*) is the name given to a pain which is confined to one spot and occurs without any known cause; *causalgia* (*Weir Mitchell*) is the name given to a burning sensation of pain. Many patients give a history of pain when they have simply feelings of malaise or irritability. The localization of pain in diseases of internal organs seems to have a definite relationship to the spinal innervation of the organ. Inasmuch as the sympathetic nerves of every organ belong to a specific segment of the spinal cord, diseases of this organ produce pain and hyperesthesia in that area of the skin which receives its sensory fibres from the same segment of the spinal cord (*Head*). Paresthesia is of many forms; the most common being crawling, tickling, creeping, tingling, numbness, etc. There is also a paresthesia of the temperature sense (for instance, a painful sense of cold,—psychroesthesia). The detection of paresthesia is of especial importance, as it so often calls attention to objective symptoms. Exactly where formication or numbness is felt is the location of the decreased sensation. Not rarely the paresthesia is arranged in definite nerve areas, and its borders are mapped off sharply, especially in lesions of the peripheral nerves. Sometimes the paresthesia is characterized as painful, as painful crawling, dead pain, etc. From the manner of relating the symptoms one is often able to satisfy himself of a psychogenic hypochondriacal cause for the trouble,—*e.g.*, simple sensation is not described, but rather ideas, as, "It seems as if a ball ascends from my stomach to my neck," "As if worms were crawling over my scalp," etc.

We speak of *hyperesthesia*, *hypesthesia*, and *anesthesia*.

Hyperesthesia is rare, and is not of as much clinical importance as is decrease or loss of sensation. Hyperesthesia is said to be present when painful excitation produces greater pain than in normal persons, or when an excitation that in normal persons only produces a sense of touch, pressure, or temperature, causes pain. We speak of relative hyperesthesia when stimulation below a certain area is not felt at first, then immediately after becomes painful (*Leyden*).

Often, not always, the hyperesthesia shows itself objectively by a corresponding increase of the reflexes. Often pressure upon the painful spot produces an extraordinary increase in the frequency of the pulse (*Mannkopf's symptom*).

Hypesthesia occurs oftener than anesthesia; when only slight sensation is experienced, it is generally called anesthesia. Both may exist in all the different senses of feeling (total anesthesia) or only in a few (partial). We speak of *partial loss of feeling* when either the pain sense or the temperature sense exclusively or both qualities are decreased or lost. It can occur that the feeling for warmth or cold alone is lost.

Loss of pain = *analgesia*.

Loss of touch = *tactile anesthesia*.

Loss of temperature = *thermanesthesia*.

We speak of *analgesia dolorosa* when an anesthesic area is the seat of spontaneously-arising pain. This occurs very often.

Confusion of the form of excitability, as heat with cold, is called *perversion of sensation*. It may also occur with normal persons, in whom intense cold applied only a second may cause a feeling of *heat*. Applications of very hot substances may, in the same way, be characterized as pricking; and a needle-prick may produce a feeling of burning.

A patient may suffer contact with a burning-hot substance a long time. This is used as an indication for the temperature sense. In this respect variations occur with normal people: persons working near heat, as in mills, and who often carry hot substances, can bear great heat on their hands; some perfectly healthy people can hold their fingers a certain length of time in a flame without feeling especial pain. Scars have lost all sense of temperature. In compression of a nerve the feeling for cold is first lost.

Anesthesias of movement and position are not rarely found alone. In mild cases, only slight changes (toes, fingers) are not felt or are wrongly indicated; in severe cases, the patient has no idea of the position of his limbs, and is unable to grasp the affected limb with his sound hand when his eyes are closed.

Anesthesia for one sense may be combined with hyperesthesia for another. It occurs that pricking with a needle may not be painful, while mere touch may cause pain. The opposite—tactile anesthesia combined with hyperalgesia—is more often observed. Paradoxical as it may seem, a kind of mixture of anesthesia and hyperesthesia sometimes occurs: pricking with a pin may cause a "dull" pain, yet be felt more strongly than on the sound side.

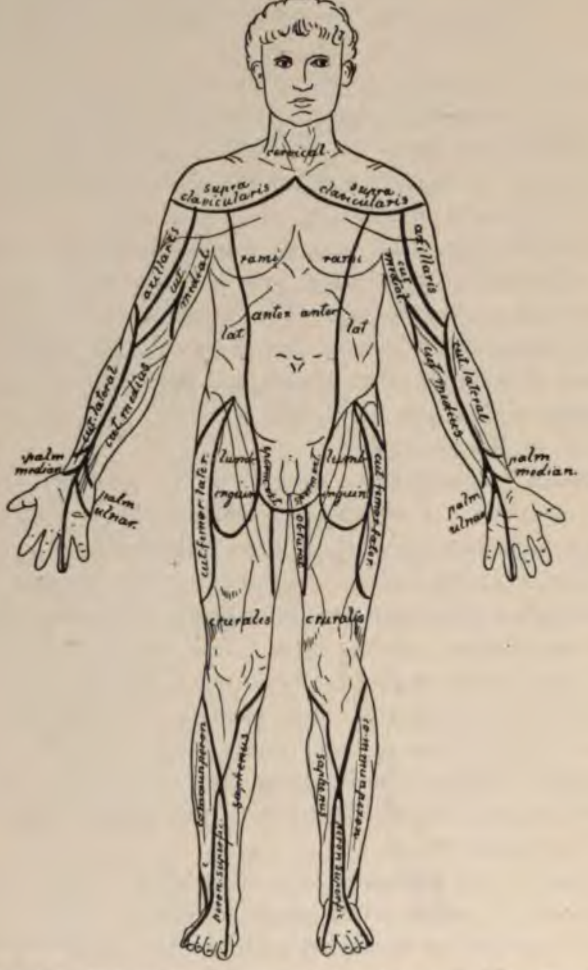
In addition to quantitative alterations, the *rapidity of conduction* should be observed. A slowness of conductivity is found in certain pathological conditions, especially as regards the feeling of pain. A prick with a needle may only become painful two to five seconds afterwards. *Double impressions* also occur, one immediately connoting the touch, and a delayed one of pain occurring later. It is rare for both sensations to be of the same degreee or that the latter one is the less painful.

Polyesthesia occurs rarely. This refers to those conditions in which simple contact is felt two or three times: when the patient is touched with a pointed substance, he imagines that two or three have touched him. *Macroesthesia,* in which substances felt appear larger than they are, is still more rare.

Perception in one extremity of a stimulation in the corresponding spot of the other is called *allocheiria* or *allesthesia* (Obersteiner). Most cases of this are due to misunderstanding,—the patient notices a paresthesia of the right leg at the time his left is being stimulated, and the objective feeling is overshadowed by the other. A real continued allocheiria has never been observed by me. In a case written up by Stewart the excitation was on the ulnar side, but was felt on the radial.

A decrease of sensation is more common than total loss: its detection and the determination of its borders may cause much difficulty. Only where there is complete absence of feeling is it easy to establish a limit between the sensitive and non-sensitive districts. Do not imagine that this is always an exact and unchangeable line. The manner of examination and the attention given by the patient at the different sittings may cause variability in the results.

FIG. 24.



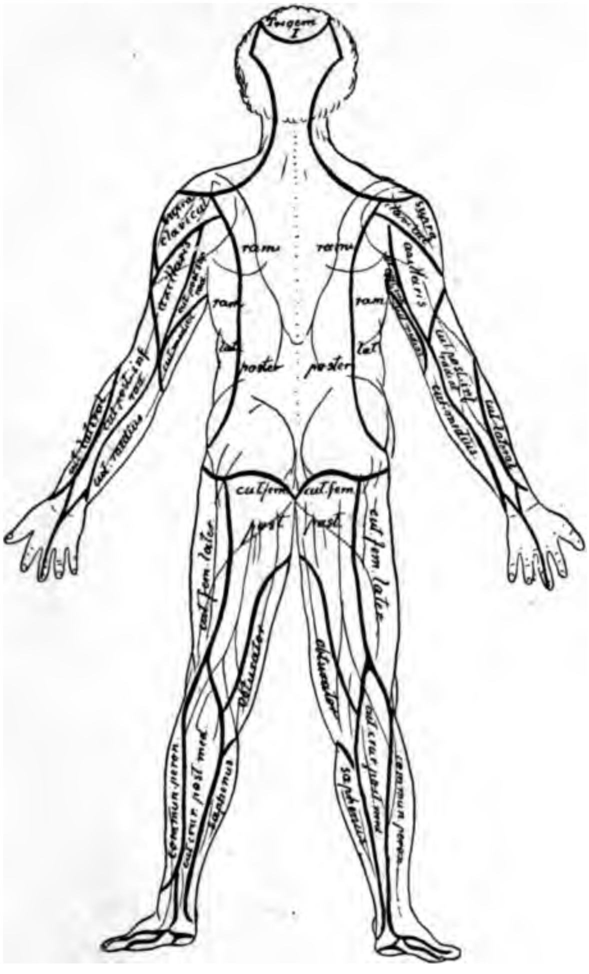
Figs. 24-31. Distribution of the sensory nerves of the skin. (After Freund.)

We may proceed by drawing the brush or needle from the sound part over the skin to the hyperesthetic or anesthetic zone, the patient telling where the sensation stops or becomes less.

The opposite may also be done. An exact knowledge of the innervation of the skin is demanded for the proper understanding of the extent of a disorder of sensation. This can be gained by a study of Freund's figures (Figs. 24–31; Fig. 26 after Frohse).

Do not forget that the zones for the different nerves vary a great deal; the borders especially are very variable. This has been particularly observed in the sensory nerves of the head by Frohse and Zander.

FIG. 25.



Only in the median regions of the head is the innervation constant; on the sides it is very variable, so that there is hardly a spot which is supplied in all individuals by the same nerve roots. A piece of skin two centimetres above the right eye may be innervated by the first branch of the trigeminal, the first and second branches, by the third, or by the third and first. In the same way the areas of the auricularis magnus, auriculotemporal, the right auricular, and vagi intermingle. In other regions, also,—on the back of the foot, the territory of the superficial peroneal, saphenous major, and anastomoticus,—variability occurs. It is noteworthy that in the face the superficial nerves of both sides extend over the middle line, so that the median area is taken care of from both sides. Zander believes this is the case also for other parts of the body. Fig. 26, after Frohse, shows the sensory innervation of the head.

FIG. 26.

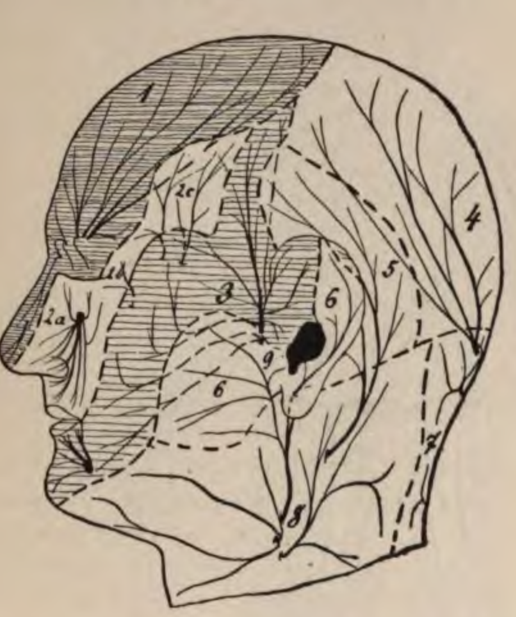


FIG. 27.

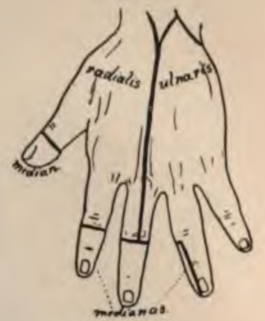
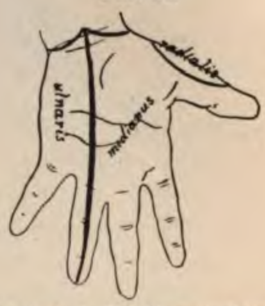


FIG. 28.



1. First branch of the trigeminal. 2. Second branch of the trigeminal. 3. Third branch of the trigeminal. Area of the first and third branches of the trigeminal marked by parellel lines; area of the auricular ramus of the vagus in the concha of the ear marked black. 2*a*, infraorbital; 2*b*, zygomatico-facial; 2*c*, zygomatico-temporal; 3, auriculo-temporal; 4, occipital magnus; 5, occipital minor; 6, auriculo-magnus; 7, cervical post. (dorsal); 8, cervical lateral (ventral); 9, auricul. vagi.

SKIN REFLEXES.

Although peripheral reflexes can be elicited in every part of the body, the plantar, the abdominal, and the cremasteric reflexes have shown themselves particularly valuable for diagnostic purposes.

We understand by the *plantar reflex* the phenomenon in which excitation of the sole of the foot produces a movement in which the foot is suddenly drawn back. Normally every form of excitation may produce this movement,—touch, hot or cold applications, tickling, etc. The stronger the stimulation, the stronger the reflex. The common form is a dorsal flexion of the foot and toes; a plantar flexion occurs rarely. A contraction of the tensor fasciæ latæ may be produced by the excitation of the sole.

In extreme cases not only the foot but the whole limb may be drawn back by flexion at the hip and knee.

The intensity of the reflex varies. Many persons show a response only to very strong excitation, as deep pricking with a needle.

Considerable variation exists among individuals as to their sensitiveness to tickling and the reflex produced thereby. We can increase the intensity of a reflex by prolongation of the stimulation or rapid repetition of single excitations. It is not necessary to test the reflex with all kinds of stimulation; it is sufficient to satisfy yourself whether the plantar reflex follows tickling or pricking of the sole of the foot. When, following slight stimulation, not merely a simple dorsal flexion of the foot but a strong jerking back of the leg results, we have an increased reflex. Notice especially whether dorsal flexion is hindered or prevented by paralysis of the extensor; in such cases the limb is always drawn back by flexion of the hip and knee.

FIG. 29.

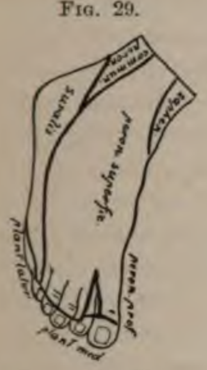


FIG. 30.

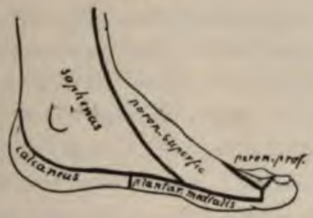
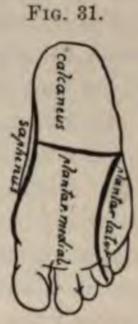


FIG. 31.



Test the *abdominal reflex* by rapidly drawing the finger or a blunt instrument over the skin of the abdominal or hypochondrial region. There ensues a retraction of the abdominal region from contraction of the abdominal muscles. This reflex is inconstant in normal individuals, and is absent altogether when the abdominal walls are flaccid or are abnormally fat. It may at times be present and at other times absent in the same individual. Its absence is, therefore, not of much pathological interest, though unilateral absence of long duration is probably always of pathological origin.

The *cremasteric reflex*[1] is produced by stimulating the inner side of the thigh with the finger or a sharp substance (in the region of the adductors). Generally such a stimulation results in a contraction of the cremaster which lifts up the scrotum. This reflex, is, however, very uncertain, as the cremaster is often found contracted and the scrotum elevated. Also, varicocele and other conditions may produce a disturbance of this reflex.

---

[1] An equivalent of the cremasteric reflex is found, according to Geigel, in women. Irritate the inner surface of the thigh, and there follows a contraction of the lower bundle of the obliquus internus.

The goose-skin or pilomotor reflex, and the gluteal, scapular, etc., reflexes are of little diagnostic importance.

The cremasteric reflex should not be confused with the *scrotal reflex*,—a contraction of the tunica dartos, with contraction of the skin of the scrotum, caused by touching or stroking it or the neighboring skin.

In testing the skin reflexes it is well to bear in mind that many may be prevented by exercise of the will-power. Such concentration of the attention is, however, necessary that the deception may be recognized by repeated examinations. Inhibition of the plantar reflex is recognized from the continued active contraction of the calf-muscles and flexors of the toes, which prevents extension. The skin reflexes have nothing in common with the deep reflexes; their actions should not be confused.

The relations between sensibility and reflex excitability are not constant: the latter may be lost with intact sensibility; it can disappear, remain, or increase, sensation being lost at the same time. This will be discussed minutely later on; at present we merely desire to present the following facts. The skin reflexes are absent with anesthesia and paralysis of spinal origin; in spinal diseases they vary according as to whether the reflex arc is disturbed or not (in the first case they are absent, in the second case present, and even increased, when the disease has developed above the region of the reflex arc. Recent observations (Bastian, Bruns, etc.) appear to prove that when conduction is totally destroyed all the reflexes, even those from the lower segment of the spinal cord, disappear.

The relations between the skin reflexes and cerebral disorders are not firmly established. We know, however, that in unilateral diseases of the cerebrum which produce hemiplegia the abdominal and cremasteric reflexes on the paralyzed side are lost. In sleep and during narcosis, as also in conditions of complete unconsciousness, these reflexes (and also the deep reflexes) are annulled.

Westphal denoted the phenomenon in which a muscle contracts when its place of insertion approximates that of another muscle as a paradoxical contraction. If, for example, the foot of a patient be pressed upward, a tonic contraction of the extensors, especially the tibialis anticus, results, which lasts some time, and which holds the foot in dorsal flexion (with adduction). The dorsal flexion resulting from tickling of the sole may also end in such a paradoxical contraction. Its meaning is unknown. It has especially been noticed in paralysis agitans and the neuroses. Of course, where the individual, believing that he should keep his foot in such a position, actively contracts his extensors, it cannot be looked upon as a paradoxical contraction.

SPASMS (HYPERKINESIS).

Spasms, in the widest acceptance of the word, denote,—(1) muscular contractions caused by unphysiological stimulation; (2) excessive muscular contractions produced by physiological stimulation. We distinguish between *tonic* and *clonic spasms*. A tonic spasm is an involuntary muscular contraction of long duration and strong intensity. Clonic spasms are those in which contraction and relaxation rapidly succeed each other. We distinguish also between *general* and local or *partial* spasms. A spasm may be confined to one muscle, one group of muscles, one limb, one part of or the whole of the body. Clonic contractions which produce marked tremulous movements of an extremity or of the whole body are called convulsions. A cramp is a tonic, painful spasm confined to one muscle or to one muscular region. Tetanic attacks are those which embrace the whole or almost the whole muscular system.

Tonic and clonic convulsions are not rarely found together.

Spasms are due to direct or indirect causes,—either to stimulations which come from the motor centres or conducting tracts, or to those which are conducted from the sensory paths to the motor centre. It is doubtful whether excitation of motor nerves can cause contraction of the muscles under their control. When in lesions and diseases of peripheral nerves a spasm is noticed in the corresponding muscles, we must bear in mind the possibility of its resulting from a reflex stimulation of the sensory branches.

The spasms may often be of reflex origin. Every painful affection, every excitation in the region of a sensory nerve, may produce spasms, and these all respond so far to *Pflüger's* law, which is, in reference to the extent of the reflex movements, that all excitations of sensory nerves affect the motor nerves upon the same side at the same heights,—a painful disease of the eye or an injury to the trigeminus produces a neuralgia of the same and not rarely a facial spasm of the same side.

Spasms are often observed which can be referred to stimulation of distant sensory nerves; for example, facial spasm resulting from uterine disorders. The so-called traumatic reflex epilepsy also comes under this class. Reflex spasms may also occur from abnormal stimulation of the reflex centres, as in strychnine-poisoning (also tetanus and hydrophobia), or when the influence of the reflex centres of inhibition is lost. Strychnine and other poisons produce such an increased excitation of the spinal cord that spasms result from the slightest stimulation.

Muscular contractions resulting from organic diseases of the spinal cord should not be called spasms in the narrower sense of the word; nor should the twitchings resulting from mere contact or percussion of the patella in cases of increased excitability be styled spasms. The distinction is, however, an artificial one.

We have certain spasms, as myoclonus, which are referable to excitation of the ganglion-cells of the anterior horns of the spinal cord. Although this is merely hypothetical, the most recent observations have shown that this is true for tetanus and strychnine intoxication. It has been surmised that spasms in the region of the motor cranial nerves may be produced by excitation of their nerve-centres. The pons and the medulla have tracts and centres which when stimulated produce convulsive movements, but never true epilepsy.

The chief centre for convulsions is the cerebral cortex. Spasms in single muscles, as well as unilateral or bilateral ones, may arise from the cortex.

The cortex is excited by organic diseases, poisons, and disturbances of circulation. It is especially, however, the functional minute lesions, which defy anatomical detection, which in the motor zone of the cortex evoke and prolong such convulsions. The spasms produced thus are recognized by the fact that emotions may increase or cause them. Concentration of the attention and auto-suggestion can also, to a certain extent, produce this phenomenon. Wundt has shown how much the innervation of the muscles of a certain part of the body is dependent upon the attention given to them.

Abnormal psychic conditions can, in addition to directly producing spasms, so influence the reflex centres that the stimulation which ordinarily only evokes reflex movements causes a spasm.

A rare phenomenon is the occurrence of spasms with rhythmical twitchings synchronous with the pulse.

VASOMOTOR, SECRETORY, AND TROPHIC DISORDERS.

The cortex of the brain contains a vasomotor centre, near the motor centres; it has been found by experiments on animals (Eulenberg-Landois). Its stimulation produces decreased temperature upon the skin of the opposite side. The tract conducting from this centre appears to go through the internal capsule. The chief centre is in the medulla oblongata, although vasomotor centres are found throughout the spinal cord. The exact locations are unknown. It has been thought that the lower central ganglion of the medulla is the chief centre. Its stimulation produces general contraction of the vascular lumen in animals. Lately Reinhold has described a large area on the floor of the fossa rhomboidalis, or fourth ventricle, as a vasomotor centre. In the spinal cord it is probably the gray matter of the anterior and lateral horns which contains the vasomotor centres. Impulses leave the spinal cord through the anterior roots to reach the sympathetic through the rami communicantes. It is not certain whether vaso-dilatory fibres are present in the posterior roots. That vasomotor fibres pass directly into the peripheral nerves is not improbable. Vasomotor disturbances may occur in diseases of almost every part of the nervous system.

Diseases of the gray matter of the spinal cord often give rise to vasomotor phenomena. The same is true of diseases of the peripheral nerves, especially the sympathetic, which receives most or all of the vascular nerves. There exist vasodilator and vasoconstrictor nerves; the last having been observed, however, only in certain places, as the chorda tympani, nervi erigentes, and sciatic.

Under *trophic disturbances* we include *muscular atrophy* and *nutritive disturbances of the skin, membranes, viscera,* and *bones.* Of the trophic disturbances of the skin we wish especially to enumerate glossy skin, canities (graying of the hair), alopecia, onychogryphosis (brittling and thickening of the nails), alopecia unguium (falling out of the nails), the development of ulcers which evince no tendency to heal (mal perforant, keratitis neuroparalytica), also many skin-diseases whose nature is not yet understood (nævi, herpes, urticaria, scleroderma, pemphigus, etc.). Charcot believed that decubitus was a trophic disorder, which other authors, however, have denied.

Spontaneous fracture (Weir Mitchell), hydrops articulorum intermittens, the arthropathies, etc.—all are classed as trophic disorders of the bones and joints. Spontaneous fracture is especially noticed in certain spinal-cord affections; also in certain developmental inhibitions of the central nervous system, but is occasionally found as an isolated phenomenon (osteopsathyrosis). These trophic disturbances are certainly varied. The physiological teachings of trophic centres and trophic nerves have silenced many theories that had previously been advanced.

The relationship between the muscular apparatus and the nervous system is the most clear. The ganglion cells in the anterior horns of the spinal cord govern the nutrition of the skeletal muscles. The ganglion cell forms a unit (neuron) with its process which passes into the anterior root and through the peripheral nerve into the muscle, where it branches. If the cell becomes diseased, the entire neuron degenerates; if the process is affected at any place, that part of the neuron degenerates which is directly connected to the cell.

The muscle degenerates simultaneously with the neuron. We do not exactly know how the ganglion cell governs the nutrition of the muscle.

The nutritive processes are probably not similar to those of functional stimulation, but occur in the same nerve-fibres, so that the conception of specific trophic nerves is not justified. Recent investigations (Marinesco, Goldscheider) show that it is not sufficient for the function of the trophic centre that it remains intact and that its connection with the muscles remains unbroken,—the excitations influencing it must pass to it from the periphery and the higher centres, for it satisfactorily to perform its duties. The preservation of the trophic centre and its connection with the muscle is, however, the important factor.

The teachings of the trophic centres for the sensory nerves are only partially confirmed. The ganglion cells of the spinal ganglia are, according to the renowned experiments of Waller, the trophic centres for the sensory peripheral nerves and those of the posterior roots and their direct prolongations. These cells comprise with the fibres named an anatomic unit, and this explains why a lesion of these cells causes nutritive disturbances of their prolongations, why a part of the nerve which is no longer connected to its cell degenerates. Marinesco's observations seem to be true in so far that the cells of the spinal ganglion to a certain extent are open to stimulation from the periphery, in order to complete their trophic function.

This does not, however, explain all pathologic alterations. The fact that, in addition to the atrophy and degeneration, *productive* changes—neoplasms and hypertrophy—are important occurrences, makes an explanation difficult. Are the spinal ganglionic cells also the trophic centres for the skin, soft tissues, osseous and articular structures, or does the nutrition of these structures depend upon innervation from certain regions of the spinal cord? Is the trophic function a specific one, or are the so-called trophic disorders of the skin, etc., only a result of an altered circulation, the decreased sensation, and injuries to the skin? Are we dealing with symptoms of degeneration, or is irritation of the nervous system a factor?

All of these questions cannot be definitely answered. Absence of sensory innervation does not appear to explain the trophic disorders (Turner, Krause). Though this renders the skin and the mucous membranes less resistant to stimulation, so that, for example, wounds heal badly, it does not explain the nutritive disorders.

It is apparent that the disturbance of circulation accompanying injury of the vasomotor nerves influences nutrition, but this circumstance cannot alone produce the manifold trophic disorders. The fact that the nutrition is supplied by the arterial nerves is one of great importance, especially when we consider that it is to a certain extent influenced reflexly by the sensory nerves. At any rate, vasomotor and trophic alterations often accompany each other. We could thereby understand why trophic disturbances accompany diseases of the peripheral nerves, the spinal ganglia, the spinal cord, and the sympathetic.

Joseph showed that extirpation of the second spinal ganglion, including the neighboring part of the anterior and posterior roots, in a cat resulted in a circumscribed loss of hair without any vasomotor phenomena.

On the whole, we can sum up the known facts and views in somewhat the following manner.

Degeneration of the muscles and nerves and their dependence upon diseases of the spinal cord need not be discussed.

Trophic disturbances in the skin, soft tissues, bones, and joints occur in diseases of the peripheral nerves, spinal ganglia, sympathetic (?), and spinal cord.

In their production, anesthesia is sometimes a factor, by rendering the structures less resistant to traumata, and by subduing pain (the "watchdog of the body").

In my opinion, however, another hypothesis may be employed to explain the pathologic alterations of a trophic character.

We assume that the function of the spinal ganglia may receive a wrong impulse, a diseased irritation; that these can only functionate normally when the stimulations from the periphery are permitted to be transmitted undisturbed to the centre. Diseases of the spinal cord which inhibit the conduction of sensory stimulation (compare the anatomico-physiological observations in the chapter upon diseases of the spinal cord) produce an accumulation of impulses in the cells of the spinal ganglion which affect injuriously its trophic function, so that an increase of nutritive changes occurs in the periphery.

In the same way, affections of the peripheral nerves, without a complete break in conduction, cause irritative conditions which are transmitted to the trophic centre and impair its function, so that nutritive changes result in the corresponding nerve area.

Anomalies of *secretion* are observed frequently. We know little concerning the perspiratory centres and tracts. It is assumed that the cortex contains centres for perspiration, that their function bears a close relationship to the gray matter of the spinal cord (anterior horns), and on the one hand that secretory fibres reach the cerebro-spinal nerves through the anterior horns, on the other hand, that they join the sympathetic. If this were true, disorders of secretion should occur in diseases of the brain, spinal cord, peripheral nerves, and sympathetic system. We find them present in such diseases, which fact points to the truth of the hypothesis.

A general hyperidrosis occurs in the general neuroses (hysteria, neurasthenia, etc.), also in exophthalmic goitre.

Excessive perspiration is often noticed on the paralyzed side in diseases of the spinal cord and peripheral nerves. Hemiplegics sometimes sweat on the paralyzed side. A *hemi-hyperidrosis unilateralis*—*i.e.*, a perspiration that occurs on one side of the face or body only—occurs in normal persons, especially after the ingestion of mustard, pickles, etc. It also occurs in diseases of the sympathetic, in hemicrania, exophthalmic goitre, erythromelalgia, tabes dorsalis, gliosis, and other nervous diseases; also as an isolated phenomenon of psychic degeneration. It may also occur as a result of an acute infectious fever.

EXAMINATION OF THE SPECIAL SENSES. DISORDERS OF FUNCTION OF THE SPECIAL SENSES.

Smell.—We must depend in testing the smell almost entirely upon the patient himself. Substances which are pungent and easily recognized and which do not stimulate the sensory nerves of the mucous membrane are held under one nostril, the other being closed. These are especially oil of peppermint, oil of lavender, oil of cloves, oil of turpentine, etc. He is asked whether he smells anything, then what he smells. If he is not able to answer, it is often due to the fact that he does not know the substance sufficiently or cannot locate its smell. It is important to compare the intensity of the sense of smell on the two sides. Here also we must depend entirely upon the intelligence of the patient. The sense of smell for ill-smelling substances is accompanied by a kind of reflex act in which the contractors of the nostril contract and the head is retracted, or the mouth is drawn as an evidence of disgust. Olfactometers have been recommended, for example, by Zwaardemaker, but clinically and practically they are of little use. Electrical testing of the smell is also not practicable at the bedside. We speak of *anosmia respiratoria* when the sense of smell is impaired by the clogging of the nostril,—*e.g.*, through inflammation, polypus, etc., and by displacements of the choanæ (*anosmia gustatoria*). Local diseases by injuring the end organs of the nerves may cause anosmia. Diseases of the olfactory nerve in its whole course may in fact cause this symptom, especially injuries to the base of the brain. Subjective sensations—hallucinations of smell—occur sometimes in diseases of the brain. A unilateral or bilateral anosmia is also not an uncommon symptom of cerebral diseases. It occurs in fracture of the base of the brain, hydrocephalus, tumor cerebri, arterio-sclerosis, focal diseases, etc. There is a senile and a congenital anosmia. The last is found in idiots and rarely also among otherwise normal persons. An hereditary anosmia has been observed by d'Abundo.

I saw anosmia occur as an isolated after-symptom of influenza and disappear again. Hyperesthesia of smell is of no diagnostic importance.

The sense of taste is tested by applying the solutions of the substances to be tasted to the tongue by means of a pipette or glass rod. The four kinds of taste, sweet, sour, salty, and bitter, should be tested with solutions of sugar, vinegar, salt, and quinine. If this is done in one examination, after each one wash out the mouth well. (Apply the quinine last, on account of its after-taste.) As the tongue and palatal mucous membranes are innervated by different nerves, it is recommended to examine each separately: (1) the anterior two-thirds of the tongue (observe especially the borders and tip); (2) the posterior part and the palato-pharyngeal region. The anterior part tastes sour substances better than bitter, while the converse is the case for the posterior. In order not to compel the patient to draw back the tongue, as is necessary in conversation, let him answer by nodding the head, or, better yet, by having him point to the proper word on a piece of paper on which "sour," "sweet," "bitter," and "salt" have been written. Among the unintelligent, little distinction is observed between salty, sour, and bitter. Taste may be influenced by every form of stomatitis or even by a coated tongue; it is absent often in old age (ageusia senilis), and unilateral or bilateral ageusia is a symptom of many nervous diseases. An electrical examination of the sense of taste can be omitted.

Hearing.—Disturbances of hearing are produced so often by diseases of the sound-conducting apparatus that an *examination* is always necessary. Functional examinations are made thus: Close one ear, and with the face turned away find at what distance a whisper or the ticking of a watch is heard. As this result is very variable according to the intensity of the whisper or tick, an examination of a normal person should be carried on at the same time. If a watch is used, determine first at what distance from the ear it is heard by a normal person. A watch is heard at a greater distance when it is gradually drawn away from the ear than if it is gradually approached. It is advisable to determine how long the vibrations of a tuning-fork are heard at a certain distance, your own ear being brought to the same distance from the fork. Special instruments (Politzer's acumeter, etc.) can be ignored, as they are not of much value.

To test the osseous conduction, tuning-forks of different tones or a loud-ticking watch are placed upon different places on the cranium, the external meatus being closed. Old people often cannot hear the ticking of a watch, as their bone-conduction is decreased.

Mechanical loss of hearing causes no impairment of such bone-conduction. It is decreased or lost in diseases of the nerves of hearing (the labyrinth or acoustic or the auditory tracts and centres). *Rinne's test* should be used to decide this. If in a normal person the vibrating tuning-fork which has been pressed against the cranium is brought in front of the ear after it has ceased to be heard, its sound can again be detected. This occurs also in nervous impairment of hearing (not, however, when it approaches deafness). In mechanical impairment of hearing Rinne's test yields a negative result.

The tuning-fork or the watch is heard through the cranial bones more distinctly with closed meatuses, because their closure converts a cavity into a resonant chamber.

A watch or tuning-fork on the frontal bone or middle of the head is heard as if from a distance, or the sound is referred to both ears. Only upon closure of the outer meatus of one ear is the sound referred to that ear. In ear-diseases which affect the sound-conducting apparatus the sound in this test (Weber's) is referred to the diseased ear. In nervous impairment of hearing it is referred to the sound ear. Schwabach claims that the fork when set on the head is heard longer than normal in middle-ear affections, less than normal in cerebral impairment of hearing. A loss of certain components of a tone-series is also regarded as a sign of nervous difficulty in hearing. Some authors consider that it is at first the higher tones which are not heard. These criteria are, however, of uncertain value, the diagnosis of nervous impairment of hearing resting on very uncertain data.

When sounds are accompanied by a feeling of irritability, it is called *hyperesthesia acustica*. The hearing of a tone deeper or higher than it really is, is called *paracusis*. It becomes *diplacusis* when the sound ear hears the normal tone.

We understand by paracusis Willisii the condition in which the hearing is improved by the simultaneous action of loud noises.

The Face.—The examination of the eyes and the eyesight is so important that it should never be omitted. The optic nerve—a part of the central nervous system which passes to the periphery and can be directly observed by the eye of the physician—gives us information concerning many diseases of the central nervous system, and often serves as a guide to a diagnosis. *He who does not understand the use of an ophthalmoscope is no neurologist.* Test the eyesight in the usual manner; determine accurately the conditions of refraction and accommodation. An account of the different methods of examination would be out of place here.

Examination of *excentric sight* is of so much importance that the most essential matters concerning it will be given here. A superficial examination is made somewhat after the following manner: Place the patient with his back towards a window and have him close one eye.

Let him fix his open eye upon your hand held at the height of his eyes and about one and a half feet distant. With the other hand approach from the periphery the field of vision, and have the patient denote by "now" the instant he first sees it. By doing this in all directions the limits are sufficiently established to determine whether large areas (a half or a quadrant) are out of the field. Somewhat more accurate is a result obtained by using a pen-holder, or something similar, with a centimetre square of paper fastened to the end, instead of the hand. Of course you must be careful to see that the eye is constantly fixed straight ahead. With this you may not only outline the borders of the field of vision, but, by shaking it in front of the eye and inquiring whether it is seen indistinctly or not at all, other defects may be detected. In the same manner test the color-sense, using red, blue, and green, instead of the white paper. More exact still is the method in which the patient is placed from one and a half to two feet from a blackboard, and, his one eye being closed, the other is fixed upon a point or cross (made with chalk) and the paper moved inward from the periphery, the place where it is first seen being marked.

The use of the *perimeter* furnishes still more exact results. One eye of the patient is directed towards the point of fixation, the other being closed with the index-finger. The meridian of the perimeter is marked in sections which are numbered from ten to ninety. Move the piece of paper slowly and keep the attention of the patient concentrated. By moving the paper in small waves it is seen earlier. The results, carried on in every meridian in order, are projected upon the plane surface,—

Upper visual field for white	50°–60°
Lower visual field for white	60°–70°
Outer visual field for white	90°
Inner visual field for white	55°–65°

The visual field is not so extensive for the other colors. Blue is next, then red, then green. The field of vision is more extensive externally than internally, because the outer part of the retina is less sensitive than the inner; the projecting nose is also partly responsible.

Do not lay stress upon slight deviations from the normal, as individual peculiarities, the manner of examination, the light, intensity of color, condition of refraction, etc., are all of moment in influencing results. An examination of the field of vision in normal persons should precede abnormal determinations.

We will notice here the following disturbances of vision:

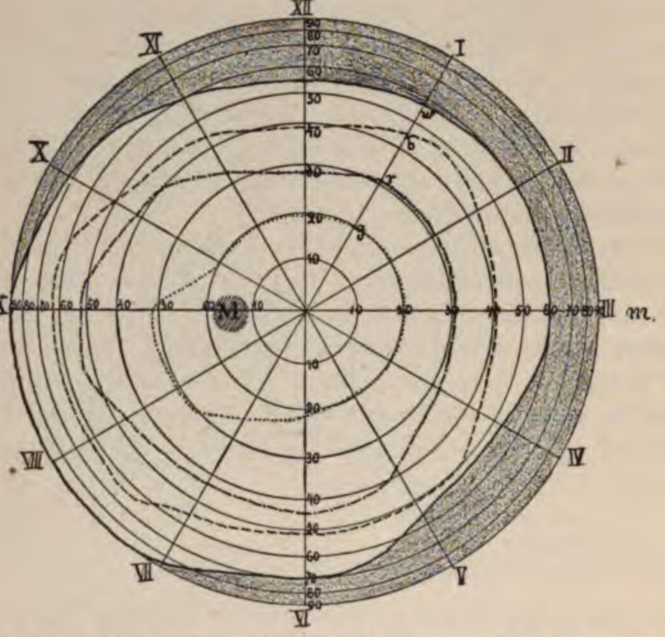
1. Concentric contraction of the visual field.
2. Scotoma.
3. Hemianopsia.

Concentric contraction of the visual field consists, as its name implies, in a contraction of the same extent on all sides. If present for white, it is narrowed for the other colors, so that the concentrically contracted vision forms, to a certain extent, a normal visual field in miniature.

A contraction of the field for colors may exist while the contraction for white is absent or slight. Myopia, myosis, and paresis of accommodation may cause slight contraction of the visual field. It may also be due to opaque corneal spots.

Examinations with Wilbrand's dark perimeter are not practicable.

FIG. 32.



Normal visual field of the left eye. (After Hirschberg.) —— Limits for white; - - - - - limits for blue; — — — — limits for red; . . . . . limits for green. M, Mariotte's blind spot; l, lateral; m, mesial side.

Scotomata are defects in the visual field,—loss of sight within the field of vision. They are often found in the region of the fixed point. Vision may not be entirely lost; may be merely relatively so. Occasionally they are present only to colors.

Hemianopsia—*i.e.*, loss of half of the visual field—is generally bilateral. If it occurs in both left or both right visual fields it is called homonymous bilateral hemianopsia (sinistra or dextra). (Figs. 33 and 34.) Loss of sight of both outer fields—*i.e.*, if the inner halves of the retinæ are defective—is called hemianopsia bitemporalis. This is rarer than the first mentioned. A superficial examination is generally sufficient for the detection of hemianopsia. In unconsciousness, aphasia, dementia, etc., it may be difficult to examine for it. With those patients whom it is difficult to get to "fix" their eyes. I proceed thus: I take two objects,—a knife and a key, or a red and a blue pencil,—bring one in front of the inner, the other in front of the outer half of the visual field, and then ask the patient what he sees. In hemianopsia he names only one object or one color.

FIG. 33.

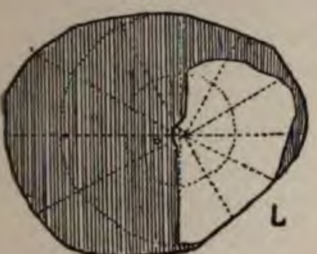
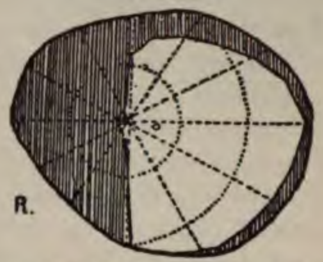


FIG. 34.



A HEMIANOPIC VISUAL FIELD.—Hemianopsia homonyma bilateralis sinistra. The outer contour shows the limits of the normal visual field for white. The shaded part represents the absent portion of the visual field. (After Gowers.)

Nasal hemianopsia is very rare, and is of no practical interest. The same is true of hemianopsia inferior and superior, the existence of which has not yet even been proved.

Electrical tests have no practical interest.

EXAMINATION OF THE REMAINING CRANIAL NERVES.

Nerves of the Eye-Muscles.—The levator palpebræ superioris lifts the upper lid and holds the eye open. When paralyzed, the upper lid droops (ptosis); if complete, the eye remains closed, or may be slightly opened by lifting the eyebrows and corrugating the forehead. By fixing the eyebrow with the fingers the ptosis appears complete again. There is also a pseudo-ptosis, caused by spasm of the orbicularis palpebrarum; its spastic character may be recognized by the tension and the resistance the lid offers to passive elevation.

The lids also contain unstriated muscle fibres innervated by the sympathetic nerves, which widen the palpebral fissure. Their paralysis causes a contraction which resembles partial ptosis, but can be distinguished by reason of the contracted pupils with normal reaction and other symptoms of sympathetic involvement. Also the patient by great will power may lift the depressed lid. A chronic contraction of these unstriated fibres and consequent dilatation of the palpebral fissure occurs also.

The Muscles which move the Eye.—The *external rectus* draws the eye outward; the *internal rectus* draws it inward; the *superior rectus* upward and inward, and at the same time turns the eye so that its vertical axis inclines at its upper end inward; the *inferior rectus* draws it downward and slightly inward; the *superior oblique* draws it downward and outward, and rotates it so that the upper end of the vertical axis is turned outward; the *inferior oblique* draws it upward and outward.

In lateral movements, both right and left, the external rectus of one eye contracts in common with the internal rectus of the other side. Normally, the eye can be moved so far laterally that the corneal border moves to the corresponding canthus of the eye. Small defects, however, are not necessarily pathological. If the lateral movement of both eyes in a certain direction is lost, we speak of *conjugate or associate paralysis of the eye-muscles.* The internal rectus, which can no longer be contracted, or but slightly, still acts during convergence (with the internal rectus of the other eye).

On the other hand, it happens that the internal rectus may still act laterally but cannot be used in movements of convergence. In "fixing" the eyes, one or both deviate outward (insufficiency of the internal recti). This occurs in errors of refraction (especially myopia) and certain nervous diseases (exophthalmic goitre, neurasthenia).

The eyeball can also be paralyzed for movements upward or downward. Paralysis of the ocular muscles causes the following phenomena:

1. *Limitation or loss of a certain movement of the eye.*
2. *Diplopia.*
3. *Secondary contracture of the antagonists.*
4. *Secondary deviation of the sound eye.*
5. *False projection of the visual field and abnormal position of the head.*

The more complete the paralysis, the greater the loss of movement. There may be also a slight paresis, which is shown by the *diplopia* that results.

Observe carefully whether the eyes can be moved freely in all directions. The weakness of an ocular muscle may sometimes be recognized from the fact that when the eye assumes the desired position it retains it only momentarily and then with slight twitchings of the eyeball.

Paralysis of an ocular muscle often produces a secondary contracture of the antagonists; for example, in paralysis of the external rectus the eye is permanently drawn inward by contracture of the internus.

The efforts of the patient to move the eye to the paralyzed side may produce an excess of innervation in the *synergist* of the other side,—*e.g.*, in paralysis of the right abducens the patient strains so much to draw the eye towards the right that the internal rectus of the left eye overacts. This causes secondary deviation of the sound eye, which appears prominently only when the affected eye is fixed. It occurs only in cases of paralytic strabismus.

Diplopia is the most important sign of muscular paralysis. The fixed eye receives the image on the macula lutea, the other receives it at some other point on the retina; hence the diplopia. In chronic cases diplopia may be absent, the patient having learned to ignore the second picture, fixing with only one eye. Very often to avoid the diplopia—which may also produce vertigo—he keeps his affected eye closed. A simple test for diplopia consists in having the patient follow your finger and tell you the moment he sees a second image. If this does not succeed use a colored (red) glass, which should be held in front of the sound eye. Now ask the patient to look at a flame or a piece of paper. If he sees a red-colored image in addition to the one of ordinary color, diplopia is present. Notice further in what part of the visual field the diplopia appears, the position of the images, and their variations in the different visual fields. The image of the normal eye is the true one, the other the false one. If the false image corresponds to the side of the eye in which it is seen, we speak of homonymous, in other cases of heteronymous diplopia. If on closing the right eye the image on the right side disappears, it is an homonymous diplopia. This is more easily recognized by testing with colored glasses. It can be regarded as the rule that convergent strabismus is accompanied by homonymous or simple, divergent strabismus by heteronymous or crossed diplopia. As the diplopia occurs first, and sometimes exclusively, in that part of the visual field to which the eye would be carried by the paralyzed muscle, the patient by assuming certain positions of the head can avoid bringing into his field of vision any part of space in which diplopia would be present. Resulting from secondary contracture, however, the diplopia may be found in the whole field.

Muscular paralysis may cause a false projection of the visual field. By reason of the strain necessary to move the eye in the direction in which the paralysis is noticed, the patient misjudges the distance, the position in space of objects, and looks beyond them. This false projection disappears if the affected eye be closed. Paralysis of an eye-muscle may be simulated by contracture of the antagonists (*e.g.*, in hysteria).

Monocular diplopia is rare. When a patient complains of diplopia—and especially when polyopia is present—examine each eye separately, the other being closed. The phenomenon results from the structure of the lens and the monochromatic aberration produced thereby (Helmholz).

Diplopia only accompanies imperfect accommodation, which may be the result of errors of refraction (myopia). In the majority of cases, however, it is *hysterical.*

If the images adjoin without any differences in height and the diplopia is homonymous, the paralyzed muscle belongs to that side to which the object must be moved to increase the distance between the images. If one image appears above the other, the false image of the affected eye will move upward in looking upward, and downward in looking downward.

Kunn has described a dissociation of eye-movements. It was inconstant and irregular, and was found in hysteria.

Paralysis of Single Muscles.—*External Rectus.*—Limitation of the external movements. Convergent strabismus, which increases through secondary contracture. Diplopia in corresponding halves of visual field of affected eye. Homonymous diplopia. The images separate when an object is moved in front of the affected eye outward. The head is turned towards the side of the paralyzed muscle.

Internal Rectus.—Imperfect movement inward. Divergent strabismus. Crossed diplopia in the same part of visual field of sound eye. Head turned in that direction.

Superior Rectus.—Loss of movement upward. In attempting to move the eye upward, the inferior oblique contracts and produces a rotation of the eye. Crossed diplopia in the upper half of visual field. The upper image, the false one, is inclined and separates from the other when the object is moved upward. Head inclined backward and towards the sound side.

Inferior Rectus.—Downward motion impaired. In attempting to look downward the eye is turned outward (superior oblique), and at the same time rotated. Diplopia in the lower half of the visual field, crossed, the false image lying deeper than the true one and moving downward when the object is lowered. The pictures are inclined. Head is bent downward and to the affected side

Inferior Oblique.—Movement upward and outward is limited. In looking upward the eye turns towards the nose Simple diplopia in the upper half of the visual field. The images are inclined.

Superior Oblique.—Slight impairment of movement. The eye is turned slightly inward upon looking downward. Simple diplopia in the lower half of the visual field; convergent strabismus. The diplopia is especially noticeable in descending stairs, the steps appearing double.

Paralysis of all the extrinsic muscles of the eyeball causes it to be immovable and to look straight ahead, and a slight *exophthalmos* becomes apparent. In paralysis of all except the superior oblique and the external rectus the eyes look downward and outward. Looking downward causes the upper end of the vertical meridian to incline inward. Diplopia in the whole field of vision.

Exophthalmos, or undue prominence of the eyes, may exist to such an extent as to prevent the lids from closing over the protruding eyeball. It is a common symptom of exophthalmic goitre. Slight degrees are also noticeable in increased intracranial pressure, especially hydrocephalus. Aneurisms, abscesses growing from behind, etc., may also push the eyeball forward. In such cases it is generally unilateral. An intermittent form occurring only in stooping has been observed, and has been supposed (Vossius) to be due to a varicose dilatation of the retrobulbar veins.

Paralysis of convergence and divergence has been occasionally noticed (Parinaud). In the first convergence is faulty or absent. Every attempt at convergence fails or is incomplete, while the synchronous lateral movements of the eyeballs are normal. Crossed paralysis exists with average-sized almost equidistant images in all directions. Occasionally they may approach upon lateral movements of the object.

The diplopia may disappear at a distance of from four to five metres. Accommodation may be normal or lessened; the pupillary reflex for convergence may be impaired while the reaction to light is normal.

More rare and difficult to test is the paralysis resulting in divergence. The patient may converge the eyeballs for near objects, but cannot bring the visual axes in parellel positions, nor fix them at all at a distance of one or two metres. The images in the median plane appear only at a definite distance away; are homonymous at the lesser distance, and are present in the whole field.

Paralysis of convergence and paralysis of divergence may occur together. Vertigo is a symptom of this form.

Conjugate deviation of the eyes may be caused by paralysis as well as by spasm of the eye-muscles. The head generally inclines at the same time. It depends either upon a tonic contraction of the associated muscles, which draw the bulbi to one side, or upon a paralysis of the antagonists.

Nystagmus denotes involuntary oscillations of the bulbi, which may take place when the eye is at rest or may appear only upon moving the eyeball. They occur in the horizontal and vertical movements especially; a rotary nystagmus is rare.

A false nystagmus is sometimes noticed, and consists of irregular jerks of larger oscillations than those of true nystagmus.

The functions of the inner eye-muscles are always to be examined; they may show the first indications of the disease.

The *sphincter pupillæ* (oculomotor) is the contractor, the *dilator pupillæ* (sympathetic) the dilator of the pupil. Contraction of the pupil may occur physiologically under three conditions:

1. Illumination of the retina (light reflex).
2. Convergence of the eyes.
3. Accommodation for near objects.

1. Suddenly throw light into a darkened eye and its pupil will contract. This contraction also occurs consensually,—that is, when the left eye is illuminated, not only the left pupil but also the right will contract. Place the patient in front of a window, close one eye, and cover the other with your hand. By rapidly removing it you allow the light to enter, and the pupil contracts. When necessary a lens or a lam may be used.

A good method is to approach a burning match suddenly from behind in front of the open eye.

This is a reflex action. The stimulation of the retina is conducted by the optic tract to the oculomotor nerve, and reaches thence the sphincter pupillæ from the oculomotor through the nucleus. We can readily see, then, how easily this reflex may be influenced by disease processes at different places. If the light reflex of the pupil is absent, we speak of reflex rigidity of the pupil. It is incomplete and atonic when it reacts slowly and slightly with intense illumination, or the contraction takes place in only a part of the pupillary region. Much practice is necessary to judge these conditions. When small pupils are normal to the individual, we must be very cautious in pronouncing a result. Guddon, Retzius, Monakow, Heddaeus, etc., have made observations which appear to show that the optic tracts do not in the strictest sense contain identical fibres for the conduction of the pupillary stimulation. The fibres probably partially decussate in the optic chiasm. Their further course is unsettled. It appears that in rare cases of disease of the optic nerves these fibres may retain their function.

In unilateral affections of the pupillary reflex the pupil does not contract to light, applied either to it or to the other eye, while the latter reacts directly and consensually. There are also cases in which the direct reaction of one with the consensual reaction of the other eye is absent,—a condition which has inaptly been called "reflex deafness."

Saenger has observed that eyes which do not react may again react under certain conditions after a long stay in darkness.

2. Convergence, the synergetical contraction of the internal recti, is

accompanied by a contraction of the pupil. It is an associated movement,—*i.e.*, the one movement is necessarily joined to the other: the volitional impulse which contracts the internal recti at the same time contracts the sphincter iridis.

This contraction during convergence is often present when the reaction to light is missing. For this reason, in testing for the light reflex, the patient should look into space and avoid every converging movement.

3. The accommodation for near objects is intimately associated with movements of convergence. But as accommodation may be normal with paralysis of the internal recti, there exists an accommodation-contraction without convergence. To examine for it, let the patient look into space, then at the tip of his nose. The reaction to accommodation may be so active as to hinder the detection of the existing loss of the light reflex. This is then only possible by keeping the patient staring continually into distance.

A new hypothesis which makes the pupillary contraction in accommodation due to the corpus ciliare pressing its blood into the iris a d by congestion of the latter producing the contraction (Knies) does not appear to us to have sufficient basis for acceptance.

Isolated cases have been described in which the pupils appear to be dilated from the action of light (*paradoxical* pupillary reaction). It was probably due to a mistake, in that it was a secondary dilatation following upon a sudden and unnoticed contraction, or that the pupil, unresponsive to light, dilated for the reason that at the moment of illumination the bulbi were fixed in a position of divergence.

Strong sensory stimulation produces dilatation of the pupils. Stick a needle into or apply a faradic brush to the skin of the neck or nuchæ, and this will be observed. It has as yet no diagnostic importance. The size of the pupil is of importance. It varies normally very considerably, but a certain variation is regarded as pathological. Pin-point pupils (myosis) are rarely normal, and are caused by poisons (especially morphine) or some disease of the nervous system.

In the aged the pupils are often small and react slowly. Increased width (dilatation) depends upon stimulation of the sympathetic—*i.e.*, spasm of the dilator pupillæ—or upon paralysis of the sphincter pupillæ. It cannot always be definitely settled which is at fault. Psychical emotion and strong muscular exertion may influence it. Some pupils dilate at the mere thought of darkness or in pain.

Bechterew has observed a case of voluntary pupil-dilatation. Probably the sympathetic is occasionally under the influence of the will. Atropine produces maximal dilatation (mydriasis) by paralyzing the sphincter and stimulating the dilator. Duboisine and hyoscyamine act similarly. Cocaine dilates the pupils, but for a shorter time and to a less degree.

Eserine promotes maximal contraction by stimulation of the sphincter and paralysis of the dilator. Morphine contracts the pupil, causing it to react slowly to light. Paralysis of the sphincter iridis produces wide, fixed pupils (mydriasis paralytica). Stimulation of the sphincter produces a contracted pupil (myosis spastica) with impairment of the reaction. Paralysis of the dilator causes a slightly contracted pupil (myosis paralytica), but it contracts fully to light.

Spasm of the dilator produces dilation of the pupil (mydriasis spastica), but it reacts to light, as a rule. Paralysis of both sphincter and dilator produces an average wide, fixed pupil.

Inequality of pupils (anisocoria) is an important symptom. No stress is to be laid upon slight differences; but marked variations are always pathological. They may be produced by difference in the refractive condition of the two eyes. Where this does not occur and no eye-disease is present, the symptom denotes a lesion of the nervous system.

Paralysis of the muscles of accommodation shows itself by the inability of the eye to accommodate for near objects, to recognize small objects, to read small print; but distant vision is unimpaired and a convex lens will enable one to see near objects. Myopia may entirely conceal a defect of accommodation.

FACIAL MUSCLES.

Those muscles which are innervated by the facial nerve are rarely paralyzed except together, and therefore their disturbances will be left to the chapter on facial paralysis.

SENSORY AREAS OF THE FACE.

The trigeminal innervates the cutaneous areas shown in Fig. 26. The cornea, conjunctiva, nasal mucous membrane, antrum of Highmore, tongue, and mouth are also innervated by it. The most important reflexes of this area are the *conjunctival* and the *corneal*. The examination is made thus: The eyelids are separated, but only slightly and without touching the eyelashes, so that the free movement of the lid is not hampered; the conjunctiva and cornea are then touched with a small object, as the head of a needle. In normal individuals this produces a contraction of the orbicularis palpebrarum,—that is, closes the lids. The intensity of this reflex varies in different persons. The conjunctival reflex is often very weak or can be voluntarily repressed; the corneal reflex is, however, constant, and its absence is always pathological.

The *nasal reflex* produced by stimulation of the nasal mucous membrane is of little diagnostic importance. It consists in contraction of the nasal muscles and of the zygomatici, etc.

THE MASTICATORY MUSCLES (TRIGEMINAL NERVE).

The *masseter* muscle and the *temporal* draw the lower towards the upper jaw, and act in bilateral closure of the jaws.

The pterygoid muscles produce the lateral movements of the lower jaw. In unilateral contraction of this muscle the lower jaw is pulled towards the opposite side.

In paralysis of these muscles on one side of the face the patient can chew only on the healthy side, and in closing the jaw the muscles contract only on this side. On opening the mouth the lower jaw inclines towards the paralyzed side. The patient cannot draw the lower jaw towards the normal side.

In bilateral paralysis of the masticatory muscles the jaw cannot be closed and mastication is hindered.

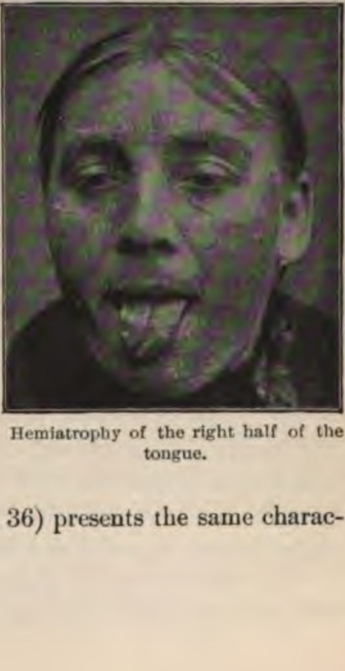
MUSCLES OF THE TONGUE (HYPOGLOSSAL NERVE).

In paralysis of the *genioglossus* on one side the tongue inclines to the opposite side.

The *styloglossus* retracts the tongue. The lingual and transverse lingual are the other muscles of the tongue. In unilateral paralysis of the tongue it inclines towards the normal side as long as it is kept on the floor of the mouth. When stretched out it inclines towards the paralyzed side.

In hemiatrophy the diseased side is decreased in size, flattened (Fig. 35), flaccid and wrinkled, tremulous, and feels doughy to the touch. Palpation often reveals this condition before it can be objectively noticed.

FIG. 35.



Hemiatrophy of the right half of the tongue.

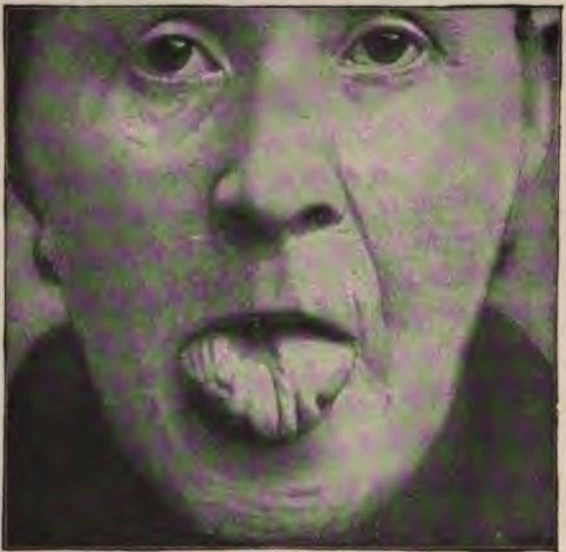
In bilateral paralysis the tongue is immovable. Bilateral paresis occurs much more often. Bilateral atrophy (Fig. 36) presents the same characteristics as hemiatrophy.

MUSCLES OF THE PALATE AND PHARYNX. DEGLUTITION.

The *levator palati* muscle elevates the velum. Its paralysis causes a dropping down of the velum upon the corresponding side; the arch formed by the free border is flatter than upon the normal side, and this difference is particularly noticeable if the patient is asked to say "ah." In bilateral paralysis the velum hangs flaccidly and cannot be elevated in phonation. The voice is markedly nasal. In drinking, a part of the fluid flows back through the nose.

The *glossostaphyline* and *pharyngopalatine* muscles close the nasal cavity from the pharyngeal side in that they draw the tongue towards the velum and the posterior palatine arch. The pharyngopalatine muscle draws the velum downward.

FIG. 36.



Atrophy of the entire tongue in bilateral paralysis of the hypoglossus nerve.

The tensor veli palatini (innervated by the trigeminal) is also called the dilatator tubæ. Its paralysis causes insufficient opening and closing of the Eustachian tube, producing difficulty in the entrance of air into the tympanic cavity and impairment of hearing. Palatal paralysis, however, was not observed by Krause after section of the third trigeminal branch. The chief nerve of the velum is the vago-accessorius. Some authors regard it as being the only nerve innervating the velum.

In order to test the functions of the palatal musculature, notice first the position of the velum during ordinary respiration and avoid exciting reflex palatal movements by pulling the extended tongue. Now let the patient phonate (say "ah" loudly). The velum will contract distinctly; the prominence formed by the uvula and the anterior palatal arch becomes steeper, though the degree of contraction is not the same in all individuals, bilateral paralysis of a slight degree being therefore difficult to recognize. It should be added that hypertrophy of the tonsils may evoke a paresis of the velum.

Speech is always impaired. It has a nasal character. A complete correspondence between the speech and the degree of visible paralysis does not always exist. The speech may be distinctly nasal, while movements of the velum in phonation may not appear to be particularly impaired.

The palatal reflex may be present or absent in paralysis of these muscles. The examination is made by touching or tickling the uvula with a sound or the handle of a spoon. A contraction of all the palatal muscles ensues.

The speech is often distinctly nasal, even though no impairment of deglutition is complained of. If the paralysis is complete, a regurgitation of fluid through the nose is almost always present, and this can be easily verified.

Some persons are naturally little sensitive to touch in this region. The absence of the reflex is not always, therefore, a positive sign of a pathological condition.

In deglutition the muscles of the lips, tongue, palate, and pharynx are brought into action. The orbicularis oris is used in the ingestion of nourishment, its paralysis causes fluids to flow out again between the lips.

The tongue in swallowing is first pressed against the hard palate with its tip, then with its back, and fluids are thereby forced into the esophagus. The tongue is also used in eating to help in forcing backward particles of food which have lodged in any part of the oral cavity.

If the tongue is paralyzed, fluids do not pass backward, but flow out again. Small particles of food remain lodged in the mouth or upon the tongue. The swallowing of food and its transmission into the stomach is the duty of the pharyngeal and esophageal muscles. Esophageal paralysis does not hinder drinking. In partial paralysis of these muscles food can only be swallowed slowly and with great exertion. In unilateral paralysis of the pharyngeal muscles, the corresponding half of the pharynx is dilated and does not contract in choking, while the other half moves in a wing-like manner.

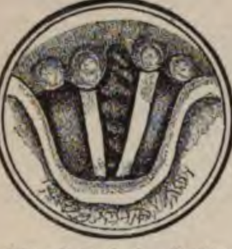
A frequent accessory symptom of deglutitory disturbances is the passage of food down the wrong channel. In drinking, the patient commences to cough, particularly when the fluid contains small particles of food. This is due to the fact that in swallowing some of the fluid passes into the larynx.

The so-called "deglutitory noises" are not of any diagnostic interest.

THE LARYNGEAL MUSCLES. DISTURBANCES OF THEIR FUNCTIONS.

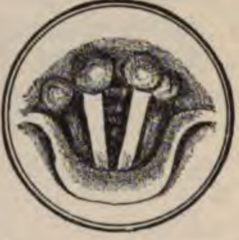
The disturbances of voice or phonation are immediately recognized by *hoarseness* or *loss of voice* (aphonia). The function of the tensors of the vocal cords may, however, be impaired without there being any noticeable alteration of speech; the vocal cord of one side may even be almost completely paralyzed without producing any pronounced disturbance of phonation.

FIG. 37.



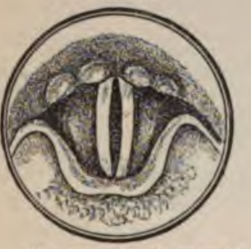
Unilateral paralysis. Position in inspiration.

FIG. 38.



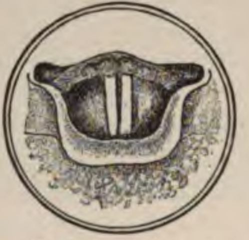
Bilateral recurrent laryngeal paralysis. Cadaveric position of the vocal cords.

FIG. 39.



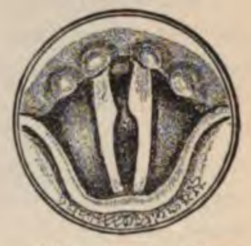
Paralysis of both internal thyro-arytenoids. Attempt at phonation.

FIG. 40.



Paralysis of both posterior crico-arytenoids. Position in inspiration.

FIG. 41.



Bilateral internal thyro-arytenoid paralysis with paresis of the arytenoid muscle.

FIGS. 37–41.—(After Strümpell and Eichhorst. Taken from Hirt's text-book.)

This shows the importance of a *laryngoscopic examination,* which is the more necessary from the fact that hoarseness and aphonia (as well as all alterations of voice-timbre) may be due to diseases of the mucous membrane and of the deeper-lying tissues, and not to muscular paralysis. A laryngoscopic examination is also the only way we have of ascertaining what muscles are involved in the paralysis. It is not in place here to explain the technique of laryngoscopic examinations.

The accompanying figures (37–41) show the laryngoscopic pictures of the most important forms of laryngeal paralysis.

Respiratory movement of the vocal cords, dilatation of the glottis, is controlled by the crico-arytenoids.

In *unilateral recurrent laryngeal paralysis* (see Fig. 37), the vocal cord of the paralyzed side is found midway between adduction and abduction (cadaveric position), and in phonation is not brought nearer to the median line. The vocal cord of the normal side extends beyond the median line and the arytenoid cartilages override each other. In inspiration the vocal cord does not move and only the normal one is adducted.

In *bilateral recurrent laryngeal paralysis* (Fig. 38), both vocal cords are found in the cadaveric position, and are not moved from this position either during phonation or respiration. Aphonia and a pronounced respiratory disturbance—inspiratory dyspnea—are present.

Paralysis of the internal thyro-arytenoids (Fig. 39) causes hoarseness. During phonation the glottis does not completely close, but an oval slit is left between the vocal cords, and these remain flaccid. If only the arytenoids are paralyzed, the glottis is closed during phonation in its anterior part; behind there remains a small triangular fissure. Combined paralysis of the arytenoids and the internal thyro-arytenoids is shown in Fig. 41.

Paralysis of the posterior *crico-arytenoids* produces inspiratory dyspnea. If the paralysis is unilateral or partial, the inspiration is noisy. An inspiratory stridor is heard, and the trouble only commences when extra demands are exerted upon the respiration. During inspiration the glottis is not dilated, but the vocal cords are drawn still more towards each other. (Something similar is noticed in normal persons when the individual inspirates forcibly; the patient must be asked to breathe quietly.)

Paralysis of the crico-thyroids causes a hoarse voice, and renders the production of high tones difficult: the laryngoscopic examination is not characteristic.

The *electric examination* of the vocal cords is possible by the insertion of an electrode into the larynx, but it is difficult to confine the stimulation to one muscle. On account of its uncertainty, one may forego it entirely. The recurrent laryngeal may, however, be stimulated in the neck, between the larynx and the inner border of the sternocleidomastoid at the height of the cricoid cartilage. Galvanic stimulation is the best; strong currents are demanded. At the moment of the closing of the current (Ca ClC.) a pronounced adduction of one or both vocal cords is noticed. The stimulation does not always succeed, particularly in fat persons with short necks.

DISORDERS OF SPEECH.

The speech may be mechanically impaired by fissures or perforations in the palate or by the loss of teeth.

More important, however, is the form which is produced by paralysis of the muscles of articulation,—*dysarthria*. Here we find that some vocals and consonants are indistinctly enunciated, and the speech is thereby indistinct and badly articulated. The labial, lingual, or palatal tones are impaired according as the lip, tongue, or palatal muscles are paralyzed. When all these muscles are involved a form of speech results that we call *bulbar*, because it occurs so often in diseases of the pons and medulla. If the speech is completely indistinct or lost, the condition is called *anarthria* (which should not be confused with aphasia). In weakness of the lip-muscles, *u* and *b*, *p*, *w*, and *f* are indistinctly formed; in weakness of the muscles of the tongue, *i* and *e* and *d*, *t*, *s*, *l*, and *r*; in weakness of the palatal muscles, *g*, *h*, *ch*, and *ng* are indistinct. *B* and *p* sound like *m-b*, *m-p*, etc.,—*i.e.*, they have a nasal tone.

Bradylalia denotes a pathological retardation of speech. In severer forms of this condition the tones are hacked and the syllables separated by considerable intervals. This is called a *scanning* speech. To detect this the patient is asked to pronounce rapidly a long word or a difficult phrase, as, *Constantinople*, *blue-bottle blackberry brandy*, *regimental artillery officer*, etc.

Stuttering is a spasmodic disturbance of speech produced by spastic muscular contractions. We have also a physical inhibition of speech caused by fear, excitement, etc. Concerning mutism and similar disorders of speech, see the special part.

Syllable-stumbling denotes the disturbance of speech in which the syllables are thrown together and certain letters introduced in the wrong places, as, "artrillery" and "artrillerary," for "artillery."

Aphasia denotes inability to understand spoken words with unimpaired hearing, or failure to translate ideas into words without there being any muscular paralysis.

II. SPECIAL PART.

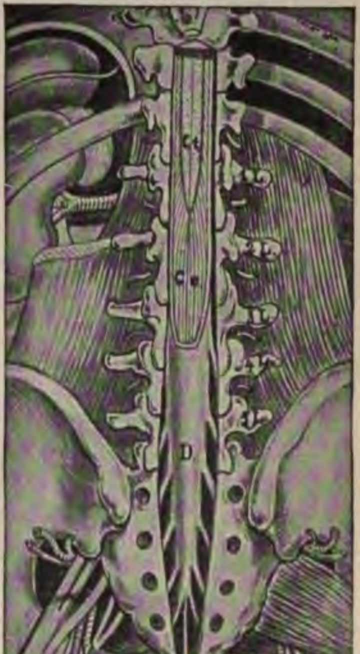
SECTION I.

DISEASES OF THE SPINAL CORD.

ANATOMY, PHYSIOLOGY, AND GENERAL PATHOLOGY OF THE SPINAL CORD.

THE spinal cord extends downward from the medulla without being sharply separated from it. The upper border is located at the origin of the first cervical nerve, the lower at the first or superior border of the second lumbar vertebra. It ends in the conus terminalis, which is still further continued downward by that complexus of nerves called the cauda equina. Injuries and diseases below the second lumbar vertebra do not, then, affect the spinal cord.

FIG. 42.



Position of the conus terminalis and of the cauda equina in the spinal and sacral canal. C. tr, conus terminalis; C e, cauda equina; 1 L., first lumbar vertebra; D, dura mater. (After Henke.)

The dura mater does not lie directly against the spinal canal, which is lined by periosteum, but is separated from it by a fatty, vascular membrane, which contains principally a venous plexus. It is also separated from the cord itself by a relatively wide space. It is thick and firm, and offers much resistance to diseases which attack the spinal cord from without.

The arachnoid is a double membrane, the outer part being attached to the inner surface of the dura, the inner to the outer surface of the pia. Many fine fibres pass from one surface to the other. Some consider only the part adjacent to the dura the arachnoid; the inner space is then the subarachnoid space containing the cerebro-spinal liquid. This space is in direct connection with the lymph paths of the peripheral nerves—whose roots receive a sheath from the dura and arachnoid—as well as with the subarachnoid space of the brain.

The pia is closely and intimately connected with the spinal cord. In addition to numerous smaller septa, it sends a thick, fibrous, and vascular prolongation into the anterior median fissure of the spinal cord, the septum medianum anticum. The circumference of the spinal cord varies at different heights; only in the dorsal region does it remain almost equal at all heights.

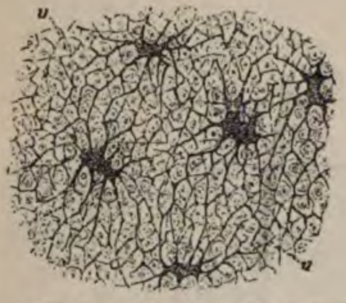
The cervical prominence begins at the third or the fourth and reaches its maximum at the fifth and sixth cervical vertebræ, and then the cord gradually decreases in volume again until at the second dorsal it passes into the dorsal portion.

The lumbar prominence is shorter and not so large. It begins at the height of the tenth dorsal vertebra, and reaches its maximum at the twelfth. The cord then decreases through the sacral part and ends in the conus terminalis.

The cauda equina surrounds the roots of the lumbar and sacral cord, the three upper lumbar roots separating from it very soon, so that the first can hardly be called a part of the cauda.

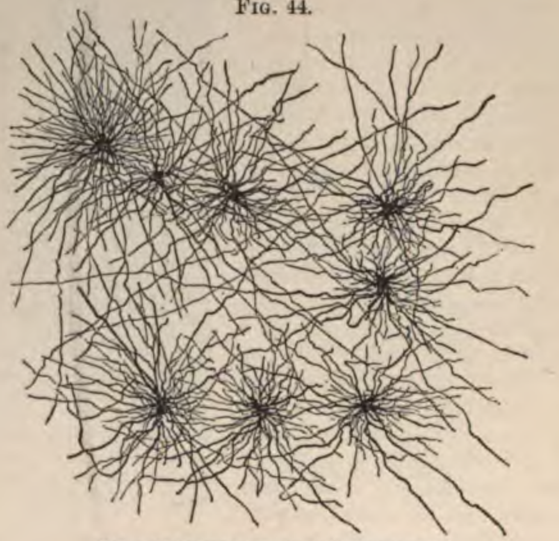
A horizontal section of the spinal cord at any height reveals that it is composed of a central gray and a peripheral white substance. The difference in color is owing to histological variability. The white substance consists of medullated nerve-fibres, which for the most part take a longitudinal course and are surrounded and enveloped by the neuroglia. A horizontal section reveals—under the microscope—numerous small circles of different sizes, which have a dark point in the centre. They are the horizontal sections of the medullated fibres, with their axis-cylinders, the intervals being filled with the neuroglia. (Fig. 43.) The neuroglia cells are bodies with a small protoplasmic area around the nucleus and with numerous arborescent processes of different lengths. (Fig. 44.)

FIG. 43.



Cross-section through the white matter of the spinal cord. (High-power microscope.) (After Kölliker.)

FIG. 44.

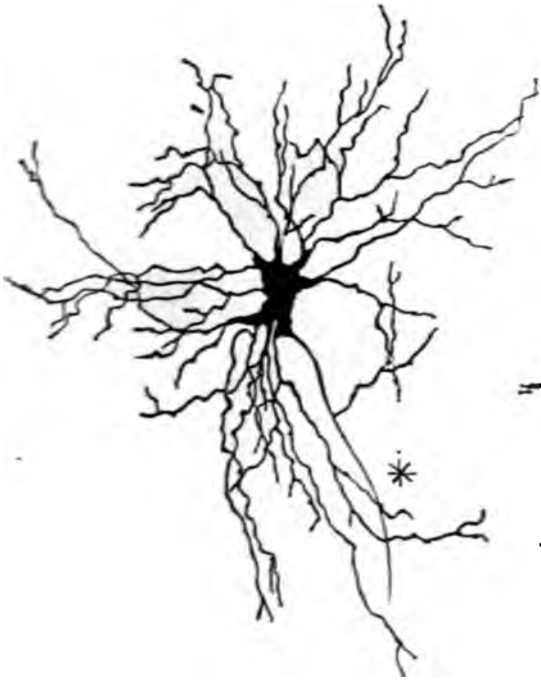


Glia cells (Golgi's type). (After Kölliker.)

Kölliker classifies them into those with long and those with short processes. In the white substance the long-process cells are especially conspicuous. The cells and their processes form sheaths for the medullated fibres and for the capillaries. The processes form a net-work or mesh which embraces the whole cord without the processes of different cells anastomosing with one another. Ranvier and Weigert regard the fibrillæ of the neuroglia as independent of the cells; Ramon y Cajal denies this. A thin covering of the neuroglia is found upon the outer surface of the cord, and in the whole periphery also; from here prolongations enter the cord.

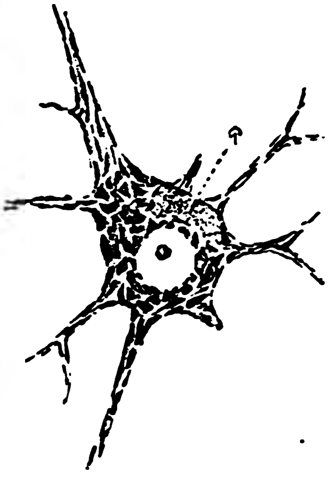
The gray matter consists of a groundwork, a thick mesh of interlacing and intertwining fibres formed by medullated nerves and their processes, the processes of the ganglion cells, and their branches, as well as the glia, which is here very richly developed. It is also more vascular than the white matter. The ganglion cells are single or grouped, but are very numerous. They possess an axis-cylinder process and numerous dendritic prolongations which branch repeatedly, and, although they end in a mesh, every process ends free (Fig. 45). According to the latest conceptions, they do not join the processes of other cells or of the nerve-fibres. The nerve-unit, consisting of the ganglion cell, its processes and collaterals, and its terminations, we call a *neuron* (Waldeyer). There exists no longer a nerve-net in the old (Gerlach's) sense,

FIG. 45.



Ganglion cells of the anterior horn with Golgi's stain. At * the axis-cylinder process. (After Lenhossek.)

FIG. 46.



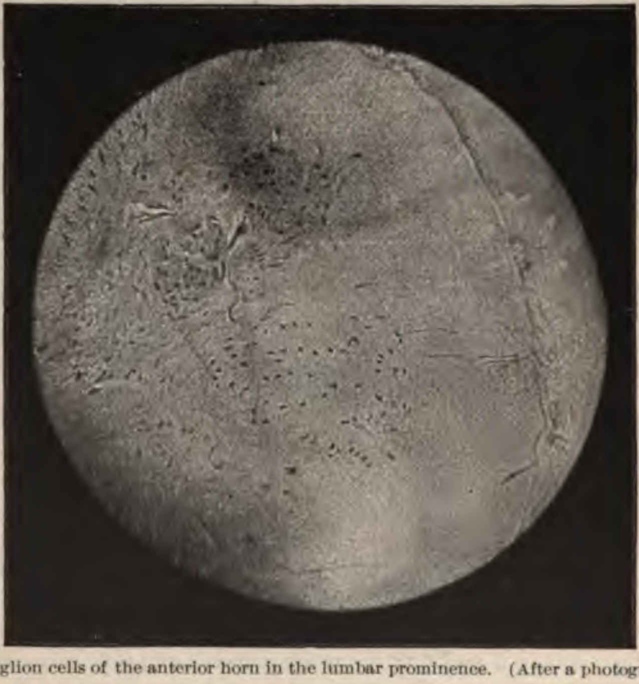
Ganglion cell with Nissl's stain. *P*, pigment. (After Edinger.)

but a nerve-mesh (neuropile). The nerve-process of most cells becomes the axis-cylinder of a medullated nerve. The lateral branches of a nerve-process are called *collaterals.* Some ganglion cells split up in the gray substance (spider cells, Golgi's cells). We have neurons of the first and second order (Waldeyer), which are also styled projection and intermediary cells.

Nissl's method will probably be of much benefit in future investigations. This method of staining permits us to distinguish the unstained basic from the colored or chromatophilous substance. The latter is found in the form of nuclei or granules—Nissl's bodies—in the cells. Their arrangement is variable. In the large cells of the spinal cord (according to Nissl, in all the motor cells) they are arranged in parallel rows. Marinesco calls the achromatic substance trophoplasm, the chromatophilous substance kinetoplasm. The physiologic significance of these different cells is so little known that it will not be considered here.

The gray substance forms the anterior and posterior horns. The anterior horns are richer in cells, and especially large cells. (Fig. 47.)

FIG. 47.



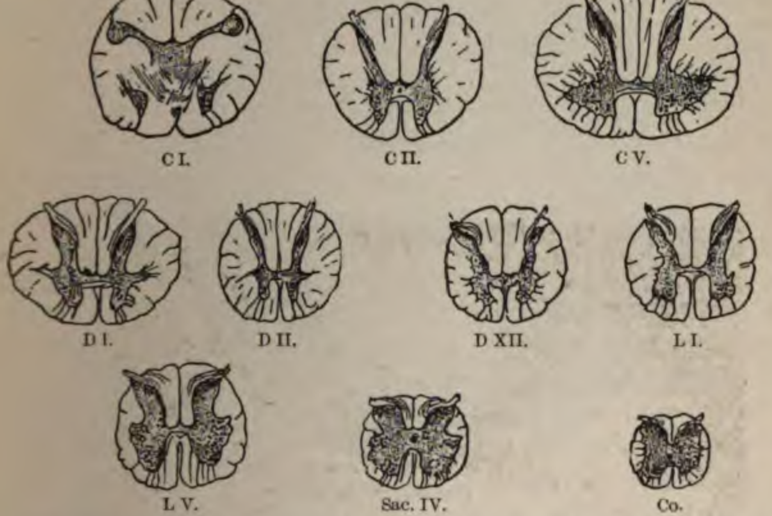
The ganglion cells of the anterior horn in the lumbar prominence. (After a photogravure.)

Some may be seen in preparations even with the naked eye. We can distinguish a mesial and a lateral group, each of which again can be subdivided into a ventral and a dorsal group. The lateral dorsal group is the most prominent. The anterior roots start for the most part from the lateral groups. The cells of the posterior horn do not form separate groups.

We find a lateral horn (tractus intermedio-lateralis) in the lower cervical and dorsal region. In the angle between the lateral horn and outer border of the posterior horn is found the processus reticuluris.

The configuration of the gray and white matter, as well as their relation to each other, varies at different heights, and enables one to recognize by mere examination of a section from what part of the cord it came.

FIG. 48.



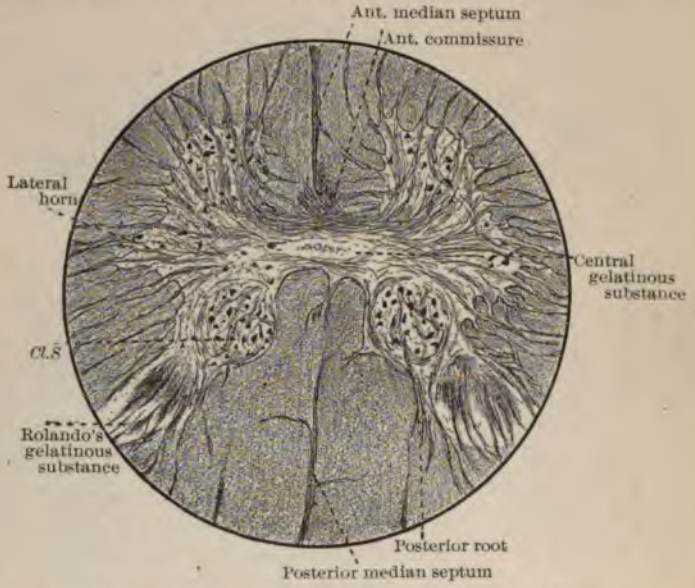
Cross-section at different heights of the spinal cord. (After Quain.) C, cervical cord; D, dorsal cord; L, lumbar cord; Sac., sacral cord; Co, conus terminalis. The Roman letters denote the pair of roots which correspond to the height of the cross-section.

Especially noticeable is the increase of gray matter in the cervical and lumbar enlargements, as well as its predominance in the sacral cord and the filum terminale. The spinal cord consists of two symmetrical halves, which are connected by the anterior white and posterior gray commissures (Fig. 49). The last surrounds the central canal, which in adults is generally occluded by a mass of cells. It is surrounded by a gelatinous substance (Stilling's subst. gelat. centralis; Kölliker's central ependymal fibres). These ependymal cells ensheathing the central canal send out long processes, which traverse the spinal-cord substance, and, at least in embryonic life, reach partly to the pia, some to the periphery.

In front both halves of the spinal cord are separated by a fissure, the anterior longitudinal fissure, into which a strong connective-tissue process of the pia forces itself. Behind they are divided by a septum (septum medianum posticum or dorsale), that is said to be formed partly by prolongations from the ependymal cells of the central canal. A posterior fissure is indicated only in the cervical cord.

The anterior roots emerge in bundles, arising in the anterior horns, and traverse the white matter; they are really processes and prolongations from the anterior ganglion cells.

FIG. 49.



Part of a cross-section through the dorsal cord. *Cl.S*, Clark's column. (After a preparation stained by Weigert's method.)

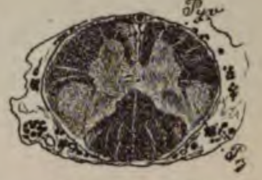
The posterior roots enter the gray substance of the posterior horn in a compact bundle, partly directly, partly indirectly; only a small number of fibres remain in the white matter.

We divide the white matter into different areas: (1) the *posterior*, that part which is bounded and surrounded by the posterior horns; (2) the *lateral*, which lies between the anterior and posterior horns; and (3) the *anterior*, which lies between the anterior horns and the anterior fissure.

The anterior and lateral divisions are not differentiated sharply from each other, as the border consists of a relatively wide region which is traversed by the anterior roots.

Developmental and pathologico-experimental studies and pathologico-anatomical observations have shown that the different parts of the white matter are not equal in development or function, but that these areas fall into a series of systems, or conducting tracts, which histologically are similar in an adult spinal cord, but which in fetal life and the first stage of development are not equally developed (Flechsig).

FIG. 50.



Cross-section through a fetal spinal cord. The crossed pyramidal and the left direct pyramidal tracts are still undeveloped; the fibres are not yet medullated. (Weigert's stain.)

In a new-born child all the fibres are medullated except those of the pyramidal tracts,—*i.e.*, the nerve-fibres of the pyramidal tract become clothed with myelin only at a later age.

We can distinguish the following tracts:

1. *Posterior Columns.*—Here we find (*a*) Goll's column (funiculus gracilis) and (*b*) Burdach's column (funiculus cuneatus). Goll's column lies closely against the septum medianum posticum; the other laterally from it, and, in the cervical region at least, separated from it by a septum (septum intermedium posticum).

2. In the *lateral areas* we find (*a*) the *lateral pyramidal* or the *crossed pyramidal tract.* It has on horizontal section a triangular shape. Only in the lumbar region does it extend to the periphery. It also does not quite extend to the gray matter. It is largest in the cervical region, decreasing downward, and is almost lost in the sacral region. (*b*) The *direct cerebellar tract.* It lies externally to the lateral *pyramidal tract*, between it and the pia, and has somewhat the shape of a quarter-moon. It is not found in the lumbar region, begins only in the lower dorsal, and from there gradually increases in size upward.

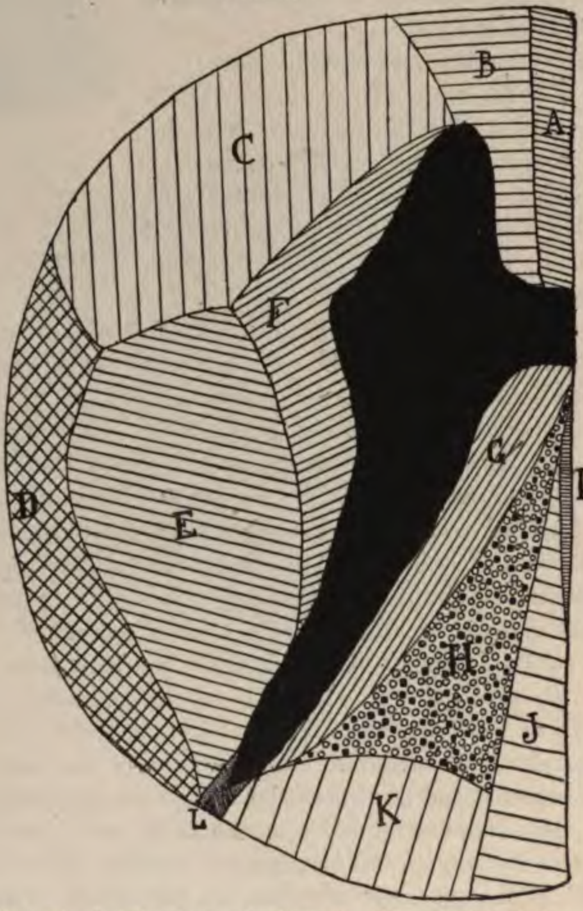
3. In the *anterior region* we find the *anterior pyramidal* or the *direct pyramidal tract.* It lies closely against the median fissure and is not very wide. Generally it is developed only in the upper cord and reaches to the middle of the dorsal region; but this is not always the case.

Less sharply mapped out is *Gowers's tract*, or the anterior lateral ascending fasciculus, and our knowledge of it is also less accurate. This tract is in the antero-lateral region, commencing on the anterior end of the direct cerebellar tract, extending somewhat farther inward, and reaching forward to the place of emergence of the anterior roots or even farther. It commences in the lumbar region. We have further in the lateral column "ground-fibres" and the "mixed lateral" zone, of which we know little.

Goll's, Burdach's, and the direct cerebellar tracts are sensory conducting paths, as is also the antero-lateral ascending fasciculus.

The pyramidal tracts are the motor tracts; they connect the motor centres of the brain with the muscles. The lateral pyramidal tracts are the crossed; the anterior are the direct motor conducting paths,—that is, the motor tract partly decussates in the lower part of the medulla oblongata, by which a greater part of the fibres passes over to the lateral pyramidal tract of the opposite side, while some fibres remain in the direct pyramidal tract of the same side. Later researches have made it probable that the last decussates in the spinal cord, so that its fibres, instead of running in a longitudinal direction, become horizontal, and reach the other side through the white commissure, whence they pass into the anterior horn.

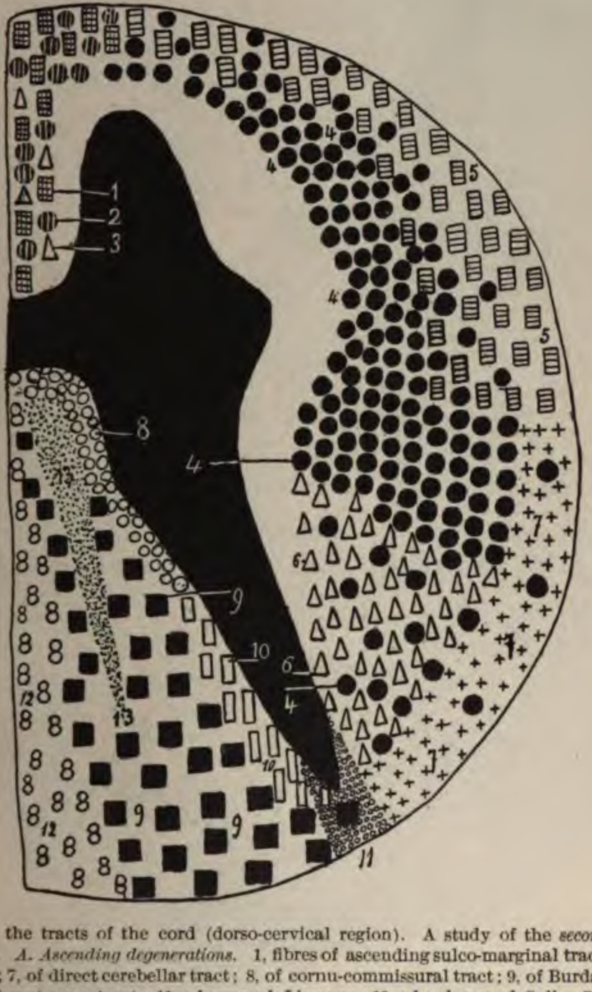
FIG. 51*a*.



Tracts of the cord. A study of their development. (After Flechsig-Marie.) *A*, direct pyramidal tract; *B*, fundamental tract of anterior column; *C*, rest of the lateral tract; *D*, direct cerebellar tract; *E*, crossed pyramidal tract; *F*, lateral limiting zone; *G*, anterior radicular zone; *H*, lesser radicular zone with two kinds of fibres; *I*, median zone; *J*, Goll's column; *K*, post.-inter. radicular zone; *L*, Lissauer's zone.

Individual variation in the above relations, absence of decussation of the pyramidal tracts, decussation of the anterior pyramidal tracts, etc., will not be discussed here. Little is known of the remainder of the white matter. It is called the antero-lateral ground bundle. Flechsig differentiates in the lateral region an anterior mixed lateral column zone and a lateral limiting layer of the gray matter, which lies close to the posterior horn, and the posterior part of the latter is again separated by him into a special fasciculus (median bundle of the lateral column). It is certain that it contains many commissural fibres which connect different heights of the cord.

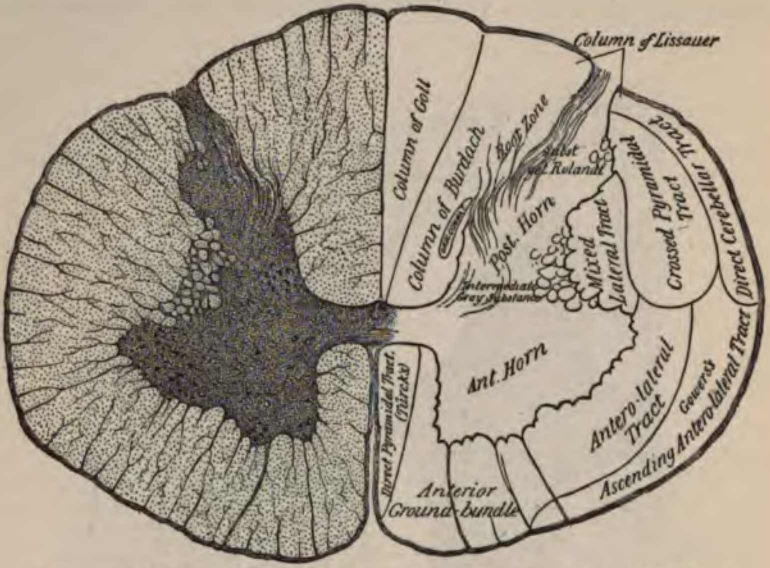
FIG. 51*b*.



Scheme of the tracts of the cord (dorso-cervical region). A study of the *secondary degenerations* (Marie). *A. Ascending degenerations.* 1, fibres of ascending sulco-marginal tract; 5, fibres of Gowers's tract; 7, of direct cerebellar tract; 8, of cornu-commissural tract; 9, of Burdach's column; 10, of external root zone tract; 11, of zone of Lissauer; 12, of column of Goll. *B. Fibres of descending degeneration.* 2, fibres of descending sulco-marginal tract; 3, of direct pyramidal tract; 6, of crossed pyramidal tract; 13, of Schultze's comma tract.

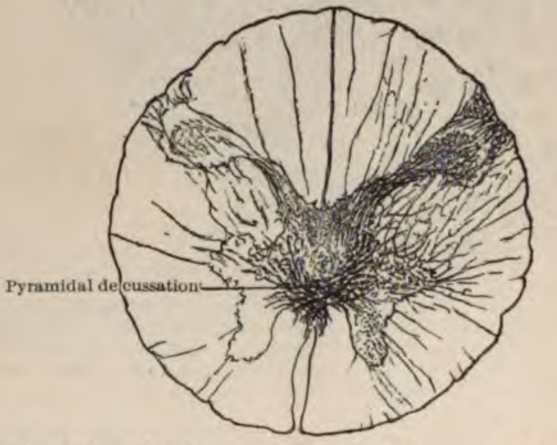
Some authors (Edinger, Bechterew, Kölliker) speak of a tract in the anterior lateral region which they consider a crossed sensory conducting tract of the second order (see below), and which is continued into the medulla. This decussation is said to take place in the anterior commissure.

FIG. 52.



Transsection of cervical spinal cord, showing its chief subdivisions. (After Mills.)

FIG. 53.



Cross-section of the cord at the commencement of the decussation of the pyramidal tract.

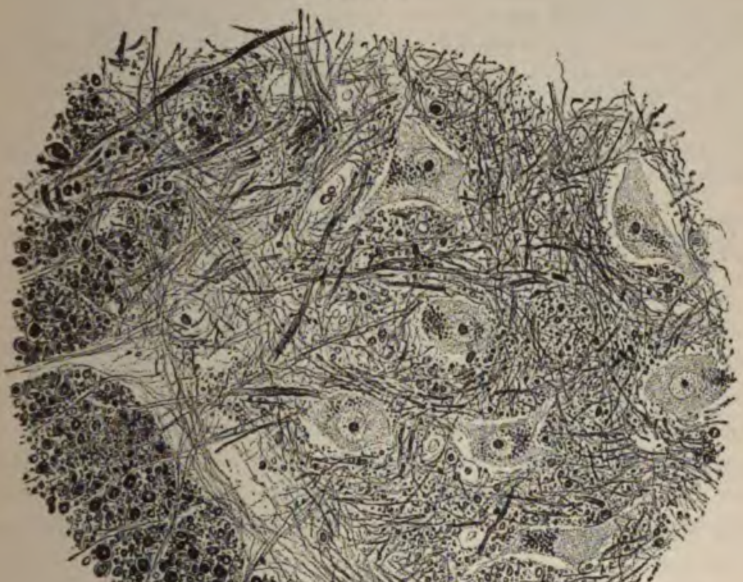
The gray matter also may be physiologically divided into the areas of the anterior and posterior horns.

The anterior horns contain the trophic centres for the muscles of the body and extremities. The impulses do not pass directly from the motor tracts to the anterior roots, but must first traverse the gray matter and the ganglion cells of the anterior horns. Disease of the anterior horns, therefore, causes not only paralysis, but also atrophy of the motor nerves and of the muscles, as they contain the trophic centres for them.

The observations of Golgi, Ramon y Cajal, Kölliker, His, etc., which, although largely made on the embryo, are nevertheless true for adults, have shown, among other things, the following:

The axis-cylinders of the pyramidal tracts give off collaterals which pass from the crossed pyramidal tracts to the anterior horns of the same side, and break up here,—*i.e.*, they end in a mesh of fibrils whose free ends adjoin the ganglion cells. (Fig. 54.) These end-brushes surround the cell without entering into a substantial connection with it. The impulses are then transmitted by contiguity.

FIG. 54.



Part of the gray matter of the anterior horn, with the neighboring portion of the lateral tract, showing the fine medullated fibres which pass from the lateral tract into the gray matter, surrounded by (pigmented) nerve-cells. (Stained by Weigert-Pal's method.) (After Kölliker.)

The anterior gray matter also transmits *reflex impulses*, among which the *tendon phenomena* must probably be included. It also contains *vaso-*

motor centres, whose location we have, however, not yet decided upon. Some place them in the lateral horns, others in the small cells of the anterior horns. We are certain, at any rate, that the stimulation passes from the gray matter to the anterior roots, to pass from there through the rami communicantes to the sympathetic. Electrical stimulation of the anterior roots with a very strong current produces arterial contraction (Pflüger).

The descending paths from the vasomotor centres of the medulla probably pass down the lateral tracts. The descending fibres in the upper cervical cord for innervation of the phrenic probably also pass down the lateral tracts (Porter). Gaskell has shown that in dogs the nerves from the second dorsal to the second lumbar, as also the second and third sacral, contain fibres for the muscles of the arteries and the internal organs,—that is, for involuntary movements.

The *cerebrospinal fibres* of the *sympathetic* are, according to Kölliker, partly sensory, partly motor. The sensory transmit the few sensations which come from the intestines. The motor transmit to the sympathetic the impulses from the cerebrospinal centres for the unstriated muscular tissue of the arterial system.

Head has attempted to establish more certain data concerning the spinal innervation of the internal organs, and has denoted certain spinal segments for certain organs, —as the first to the third dorsal segment for the heart, the first to the fifth for the lung, the sixth to the ninth for the stomach, etc. He believes that he has shown that disease of the internal organs asserts itself through pain and hyperesthesia which are localized in the corresponding segment of the posterior roots. Herpes zoster has a similar extension.

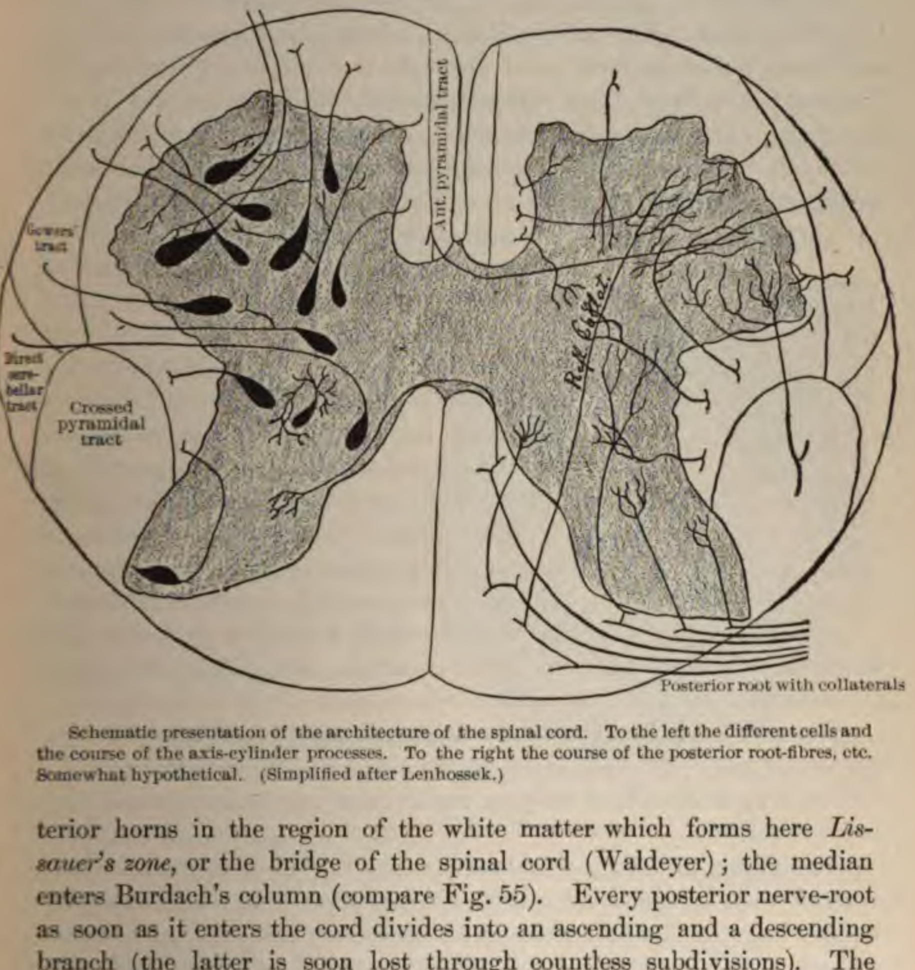
The *posterior horns* form an end-station and passage-way for the sensory tracts. Pathology shows that the *pain* sense and *temperature* sense are especially associated with this region. Reflexes are also transmitted through the posterior horn. The gray matter has probably a *trophic* influence upon the skin (probably also upon the bones and joints). It is doubtful whether there are any fibrils coming from the cells of the posterior horns which help to form the posterior columns, though many observations would seem to indicate that in the posterior commissural region (so-called ventral field of the posterior tracts) such fibres are found, while this region, according to other authors, contains the descending processes of the posterior root-fibres.

In the *dorsal region* we find between the anterior and posterior horns, in the median region of the gray matter, a well-defined group of cells called the *posterior vesicular column of Clarke*. It consists of large bipolar cells with axis-cylinder processes in the midst of bundles of fine fibres. Smaller cell groups corresponding to it are found at other heights also. We know the following facts concerning the *different tracts and their course.*

The posterior roots, at least for the most part, if not entirely, arise in the *spinal ganglia*. The cells found here are bipoplar,—they appear unipolar in adults because both processes lie beside each other; the one process goes through the posterior roots to the spinal cord, the other as a sensory nerve-fibre passes to the periphery. Recent observations seem to indicate that the posterior roots contain fibres not dependent upon the cells of the spinal ganglia, and which have their trophic centres in the periphery: in the skin, mucous membranes, or even in the spinal cord.

The posterior roots, upon entering the spinal cord, form a lateral and a strong median bundle. The lateral lies on the periphery of the posterior horns in the region of the white matter which forms here *Lissauer's zone*, or the bridge of the spinal cord (Waldeyer); the median enters Burdach's column (compare Fig. 55). Every posterior nerve-root as soon as it enters the cord divides into an ascending and a descending branch (the latter is soon lost through countless subdivisions). The ascending bundle forms Burdach's column, but in the upper parts is forced inward by the other root-fibres that enter the cord until they are pushed over into Goll's columns. The lower the sensory roots enter the more they approach the middle; so that sensory continuations of the sacral

FIG. 55.

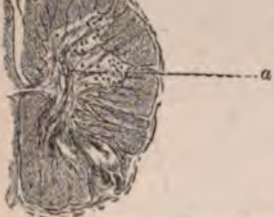


Schematic presentation of the architecture of the spinal cord. To the left the different cells and the course of the axis-cylinder processes. To the right the course of the posterior root-fibres, etc. Somewhat hypothetical. (Simplified after Lenhossek.)

roots lie in the cervical cord in the middle of Goll's columns. *Collaterals* coming from the posterior root-fibres, as well as their longitudinal branches, enter the gray matter of the posterior horns at all heights. This is especially the case in the middle third of Burdach's columns. We see, then, that part of the fibres of the posterior roots enter the gray matter directly, a part only in the upper regions of the cord, and a *third remains in the posterior tract* to sink into the gray matter in the medulla (the nuclei of the posterior columns).

In the dorsal region many of the posterior root-fibres pass into Clarke's column. The collateral fibres which enter the posterior horns and Clarke's columns form a net-work, the free ends of which surround the ganglion cells of these regions and their dendrites without entering them. The substantia gelatinosa Rolandi contains many of these collaterals. Transmission of sensory impulses from the ganglion cells to the gray matter is therefore also by contact. A part of the sensory fibres which pass into the posterior horns extend to the gray matter of the anterior horns and branch there. These are probably reflex collateral fibres. (Fig. 56.) It is also known that some of the sensory fibres or their collaterals pass from the white matter of the posterior tracts into the posterior commissure, *decussate* there, and enter the other posterior horn.

FIG. 56.



Part of a cross-section of the cord. Pal's stain. At *a* fibres which pass from the posterior root to the anterior horn. (Reflex collaterals?)

The *direct cerebellar tract* commences in Clarke's columns, its fibres being prolongations of the ganglion cells in these columns. Whether this is its only origin and whether the direct cerebellar tract is the only prolongation of the *columns of Clarke* is not known. This tract passes through the restiform body to the cerebellum.

Gowers's tract[1] is probably also a crossed sensory conducting tract of the second order; its origin is not yet definitely settled; Bechterew thinks that it arises from cells of the gray matter between the anterior and posterior horns.

In addition to the motor cells of the anterior horns from which the anterior roots arise and the cells of the posterior horns which carry the sensory impulses from the fibres of the posterior roots, the gray matter contains numerous ganglion cells which are called *commissural cells* and *column cells*. The processes of the commissural cells pass through the anterior commissure to the opposite side, and run in a longitudinal direction after reaching the anterior column. Some send their processes to the gray matter of the opposite side (Lenhossek). The column cells are subdivided by van Gehuchten into (1) *cells of the dorsal substance* on the side on which they originate; (2) cells the axis-cylinders of which pass to the white matter of the opposite side *via* the anterior commissure; (3) cells the axis-cylinders of which bifurcate in the gray substance, some going to the white matter of the same side, some to the antero-lateral columns of the opposite side. These fibres thus cause an intimate relationship between all the different heights of the spinal cord, associating them closely together.

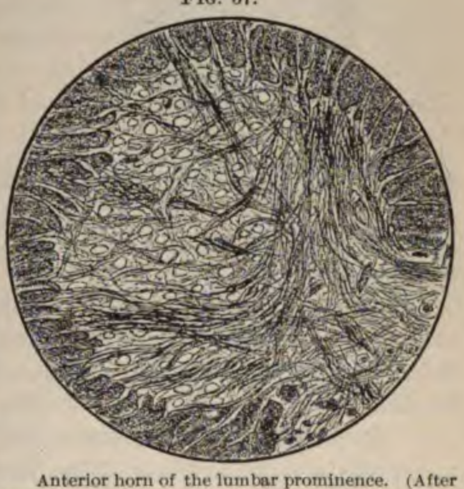
---

[1] Edinger calls this the ventral cerebellospinal fasciculus, and the direct cerebellar tract he calls the dorsal cerebellospinal fasciculus. It is improbable that fibres pass centrifugally from the cerebellum into the spinal cord.

We do not know anything definite concerning the course of those *reflex inhibitory* fibres which descend from the brain, though it is probable that they descend with the pyramidal tract (Kölliker).

The sensory tracts then, after entering the spinal cord, pass directly to the gray matter of the posterior horns, or, after some deviation, to Burdach's column. Most of the fibres enter the posterior horn of the same side; a few decussate in the posterior commissure. A part of the sensory fibres are carried by Burdach's and Goll's columns to the medulla oblongata. In the dorsal cord most of the fibres of the posterior roots, after reaching the gray matter, enter Clarke's columns; the sensory fibres found here and in the posterior horns form a network whose end brushes surround the ganglion cells and their processes. The direct cerebellar tract arises in the cells of Clarke's columns. These are, therefore, sensory conducting paths of the second order. The same is probably also true for Gowers's tract, though we as yet do not know from what cells it originates. Part of the fibres entering the gray matter reach the cells of the anterior horn and probably serve as transmitters of reflex movements, though there are longer tracts for these, the sensory excitation passing to the column cells, and from these through the processes which traverse the white matter or their collaterals, whence it passes to cells at other heights of the cord.

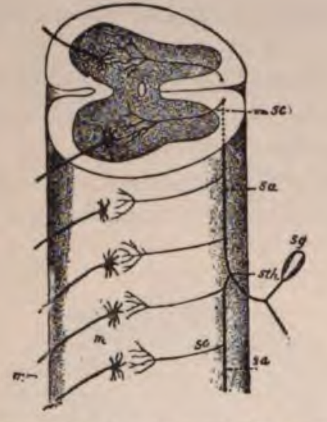
FIG. 57.



Anterior horn of the lumbar prominence. (After a specimen stained by Weigert's method. Low-power enlargement.)

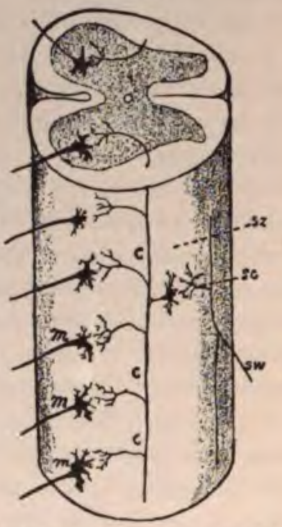
Motor impulses are conducted through the pyramidal tracts, and carried by means of their collaterals to the gray matter of the anterior horns; from there to the motor ganglion cells, whence they reach the anterior roots. The motor path consists of two neurons: the one (a direct motor neuron, or a neuron of the first class) extends from the cells of the anterior horn to the end brushes in the muscle; the second (an indirect, or neuron of the second order) extends from the cells in the motor part of the cortex to the end brushes in the anterior horn. Waldeyer calls the central an *archineuron;* the peripheral, a *teleneuron.* The sensory path consists of at least two, perhaps more, neurons. Fig. 57 shows the thick net-work of the large medullated fibres in the anterior horn.

FIG. 58.



Scheme of the elements involved in the spinal reflexes. A sensory root-fibre, *s*, connected with a cell of the spinal ganglion, *sg*, divides into an ascending branch, *sth*, and a descending one, *sa*. Both of these give off collaterals, *sc*, which act upon the motor cells, *m*; *mw*, motor root. (After Kölliker.)

FIG. 59.



A sensory root-fibre, *sw*, gives off a collateral, *sc*, which acts upon a column cell, *sz*, and through the collaterals of the fork-like dividing nerve processes of it, *c*, excites a number of motor cells, *m*. (After Kölliker.)

In every segment of the spinal cord centres for certain muscular groups, and *reflex centres* for the reflex movements in them, are found. Probably they are the same complex of ganglion cells which are called into action by the conduction of motor impulses which have descended the pyramidal tracts to the anterior roots, and are also brought into play in the transmission of reflex movements. There is also an inhibition of reflex movements.

When the skin reflexes are called into action the following route is followed. The excitation enters the posterior root, reaches the gray matter, and from there passes either directly to the neighborhood of the corresponding cells of the anterior horn through the reflex collaterals of the posterior roots or is transmitted to cells farther on. It may be either a spider cell (Golgi's cell, reflex cell) or a column cell to which the sensory impulse is first transmitted. Through the latter, by means of its processes and collaterals, it can reach different segments of the spinal cord. (Fig. 59.) It also might be carried directly to the motor cells of different segments by being transmitted up the long ascending fibres of the posterior tract (Fig. 58).

It is to be taken for granted that the reflex excitation will take the shortest path, so that simple reflexes will have their *reflex-arcs* in the same segment of the spinal cord in which the posterior root enters. Complicated reflexes, on the contrary, have a long reflex-arc, are carried to many segments, or are transmitted through the medulla oblongata. Pflüger's laws are in accord with modern conceptions and knowledge concerning the architecture of the spinal cord.

Every lesion of the reflex-arc which lowers its conductivity produces decrease or loss of reflex movement. If the excitation of the reflex centres is increased through disease, the reflexes are increased. Diseases whose seat is above the reflex-arc may increase or decrease it. If the lesion is an incomplete one, as is generally the case, the reflexes are increased. It seems from recent observations that a total destruction of conduction above the reflex-arc will decrease or annul the reflexes, though this is not yet fully proved.

What has been said concerning the skin reflexes holds true in general for the tendon reflexes, though the correspondence in different diseases is by no means complete. Concerning the reflex inhibitory fibres we do not know anything positive; perhaps the motor fibres have such functions. Voluntary innervation of the muscles in question will inhibit the reflexes.

The knee reflex is lost in the following cases:

1. In a lesion of the centripetal part of the reflex-arc in question,—as in a neuritis of the crural nerve or in an affection of the posterior roots and posterior tracts of the corresponding part of the spinal cord.
2. In disease of the gray matter at the height of the reflex-arc.
3. In disease of the centrifugal part of the arc, the anterior roots belonging to it, and the motor fibres of the crural nerve.
4. In disease of the spinal cord above the reflex-arc in which there is a complete break of continuity (?).
5. In deep coma.

An *increased knee reflex* occurs,—

1. From excitation of the centripetal part of the reflex-arc. Slight

lesions of the posterior roots in which there is no break of continuity may in this way produce an increased knee reflex.

2. From excitation of the reflex centre itself (strychnine, tetanus).

3. In diseases of the crossed pyramidal tracts (not resulting from a total break of continuity above the reflex-arc).

4. In diffuse diseases above the reflex-arc which have not produced a complete break of conduction.

5. In the functional neuroses accompanied by a general increase in excitability.

Van Gehuchten has called attention to the fact that the condition of the reflexes and knee phenomenon depends upon the "nerve-tonus" of the motor ganglion cells as well as upon the condition of the reflex-arc. This results from excitation and impulses passing from the periphery and centre. It is increased in degeneration of the pyramidal tracts and annulled in complete destruction of conduction in the spinal cord, etc.

We have still to consider those centres which are found at certain heights in the gray matter.

We find in the lower cervical and upper dorsal part of the cord, at the origin of the first dorsal root, the *ciliospinal* centre,—a centre for the muscle which dilates the pupil and the unstriated palpebral muscle whose contraction widens the palpebral fissure. Excitation of this centre, therefore, produces dilatation of the pupil and of the palpebral fissure of the same side. Its conducting path leaves the spinal cord with the *anterior root of the first dorsal nerve*, and reaches the sympathetic through the ramus communicans. Perhaps some fibres come from the eighth cervical root.

By electrical stimulation of the first dorsal root of a living person, I secured maximal dilatation of the pupils, while no result was obtained from the second.

Seguin observed myosis after section of the roots of the lower plexus.

Kocher believes that the oculopupillary fibres arise in the medulla and descend to the first dorsal root, where they leave the cord.

In the sacral segment of the cord, or in the filum terminale, we find centres for the bladder, the rectum, and the sexual apparatus. These centres regulate the evacuation of the bladder and induce ejaculation of semen. Some observations tend to show that the centre for the sexual apparatus is not found close to the ano-vesical centre.[1]

The laws which govern these functions and the manner in which

---

[1] The erigentes nervi arise from the first, second, and third sacral nerves; the fibres for the ischio-cavernous, transverse perineal, and bulbo-cavernous from the third and fourth. The sympathetic also has some influence in the vasomotor innervation of the penis.

these centres and the brain are associated are not clearly defined. We can say with certainty, however, that the gray matter of the lowest segment of the cord contains a centre for the sphincter and the detrusor vesicæ. The motor impulses pass from these centres through the anterior roots of the third and fourth sacral nerves to the common pudendal nerve, or to the hemorrhoidalis medius and the sympathetic, and then to the muscles of the bladder. Some sensory fibres pass from the mucous membrane of the bladder along the second, third, and fourth sacral nerves to the spinal cord and connect with these centres; the sensory stimulation is also conducted to the brain.

The bladder functions are carried on in the following manner. If the bladder is full, the sensory nerves are stimulated; the excitation is carried to the brain, whence an impulse goes to the centres in the spinal cord, and produces a contraction of the detrusor. We assume that there is an antagonism between the detrusor and the sphincter centre in this manner, that a stimulation of the first produces an inhibition of the latter, and thereby a relaxation of the sphincter. The will, however, regulates such action; it can contract as well as relax the sphincter vesicæ, and can prevent its reflex contraction. It does not act directly upon the detrusor; whether the hypothetical antagonism asserts itself in the voluntary inhibition of the sphincter, producing an excitation of the detrusor, is not known positively. The will may, however, produce a contraction of the abdominal muscles, and thereby promote the evacuation of the bladder. We do not know positively by which tracts impulses are carried from the brain to the bladder centre; probably by the antero-lateral tracts.

From the above it is easily perceived that disorders in function of the bladder may be due to lesions of any part of the cord. If the break in continuity lies above the bladder centre—that is, above the sacral segment—and is complete, the impulse for evacuation is lost and the will has no more influence. As soon as the bladder is full, the evacuation proceeds reflexly, the patient not being able to hold back his urine (intermittent incontinence of urine). If the centres themselves are destroyed, the sphincter is continually relaxed, the detrusor is inactive, and there results a constant trickling. The closing of the bladder may be promoted by the elasticity of its mouth, so that only upon the accumulation of large quantities of urine can an evacuation drop by drop ensue. Other factors, as the altered pressure of the internal organs, may have under such conditions an influence upon the evacuation of the bladder; pressure upon the corresponding abdominal region, for instance, will produce a partial evacuation in paralysis of the sphincter (Wagner). The purely mechanical factors should therefore always be taken into account in judging of the existence of bladder paralysis. Recent observations seem to show that in total section of the spinal cord at any height reflex evacuation of the bladder and rectum may be absent. Paralysis of the detrusor produces retention of the urine (ischuria). The bladder is enormously distended until finally the urine drips away mechanically (ischuria paradoxa). Retention of the urine may also be caused by spasm of the sphincter,—*i.e.*, through inability voluntarily to relax the sphincter. Retention of urine by reason of spasm of the sphincter is a common occurrence after a total dissolution of continuity above the vesico-spinal centre. Retention and incontinence may also result when the sensory paths alone are diseased.

The evacuation of the rectum is caused by a similar mechanism. Incontinentia alvi results from disturbance of the sphincter centre. Hard feces, by reason of the elasticity of the os, may be retained. If the conducting paths to the brain are broken, the will loses its influence upon the external anal sphincter, though reflex contraction is still possible, and may even be increased. By inserting the finger into the anus, we may feel the reflex contraction and prove that the disease exists above the centre. If conduction be totally destroyed, however, this reflex may be absent. The will has no influence upon the rectal muscles which produce defecation, though it may aid efforts of defecation by contracting the abdominal muscles.

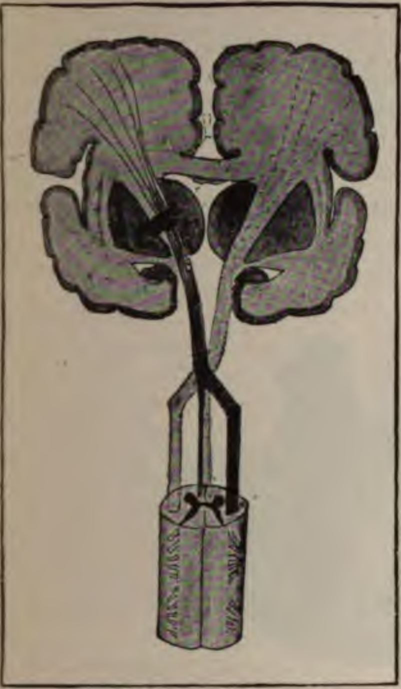
SECONDARY DEGENERATION.

The motor paths represented by the direct and crossed pyramidal tracts have their trophic centre in the cerebral cortex, in the ganglion cells of the motor zone, the fibres which run in these tracts being direct prolongations of these cells. The cells of the motor cortical area form therefore with the fibres of the pyramidal tracts a nerve unit. Separate these fibres from the cell and they become useless and atrophy.

Every disease-process, therefore, which disturbs motor conductivity at any place produces a degeneration of the section that is no longer in connection with its trophic centre, a *descending degeneration* (Türck). A degeneration of the motor path in the brain, the pons, or the medulla produces, therefore, an atrophy of the corresponding direct and crossed pyramidal tracts (Fig. 60; compare also Fig. 61). If the break takes place in the spinal cord, which occurs in injuries or diseases which disturb the cord at certain heights (transverse myelitis, compression of the spinal cord, etc.), we find a degeneration of the pyramidal tracts below the seat of the lesion. The direct pyramidal tracts take part in the degeneration only when the disease attacks the upper part of the cord, as this tract normally descends only to the middle or lower dorsal cord. Fig. 60 shows a degeneration confined to the crossed pyramidal tract.

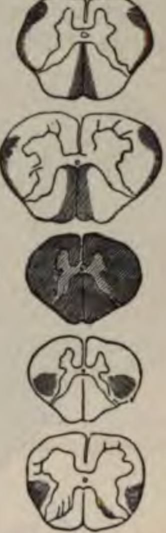
While the *motor conducting paths degenerate in a descending direction,*[1] the *secondary degeneration of the sensory paths is an as-*

FIG. 60.



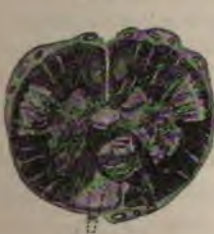
Schematic representation of the descending degeneration from a lesion in the internal capsule. (After Edinger.)

FIG. 62.



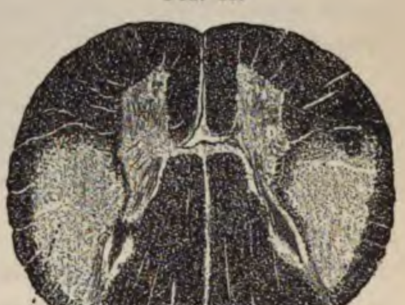
Secondary ascending and descending degeneration from a complete transverse affection of the upper dorsal cord. (After Strümpell.)

FIG. 61.



Descending degeneration of the left pyramida tract from cerebral disease. (Pal's stain.)

FIG. 63.



Degeneration of the crossed pyramidal tract from cerebral disease. (Pal's stain.)

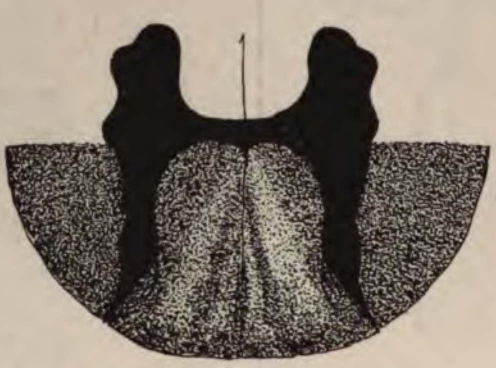
---

[1] Marie, Loewenthal, etc., have described other tracts which degenerate in a descending direction, but, as they are not sharply defined nor accurately described, we will ignore them.

cending one, as these arise in ganglion cells which are found in the spinal ganglia, in Clarke's columns (and also in the posterior horns?), and here run upward in a centripetal direction.

A section of the cord at a certain height, or a disease acting similarly, produces the following alterations above the lesion. Just above the lesion the whole *posterior column* is degenerated, as is also the direct cerebellar tract, and generally the *antero-lateral fasciculus*. The degeneration of Burdach's column soon disappears, however, as we pass upward, as this tract rebuilds itself at every new height from the entering posterior roots. In the cervical cord we find, therefore, only a degeneration of Goll's tract, of the direct cerebellar tract, and of Gowers's bundle (Fig. 62).

FIG. 64.



Dorsal cord twenty years after amputation of the left leg. Retrograde degeneration.

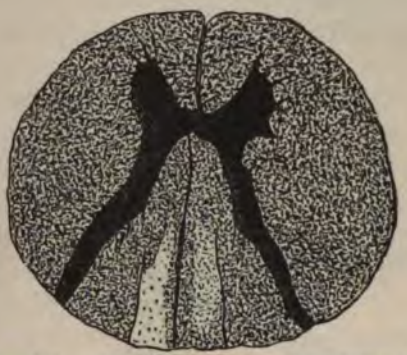
A descending degeneration of the posterior cords has been noticed, which, however, proceeds only a short distance in two small, comma-shaped areas (Schultze). (It is probably the descending branch of the sensory roots which we spoke of on page 104.) The same may be true for Flechsig's oval field, which lies near the posterior septum.

An ascending degeneration is also observed after section of the posterior roots and diseases of the same. The experiments of Schiefferdecker, Kahler, Singer, Münzer, etc., teach that the posterior tracts are the direct prolongations of the posterior roots. It has been assumed that descending degeneration of the motor tracts is shown clinically by a spastic symptom complex. Even if this is generally observed when on post-mortem examination a secondary descending degeneration is found to be present, it is still improbable that it is a direct result of this anatomical alteration.

The so-called *retrograde degeneration*, which lately has been much discussed, is a sort of atrophy which follows section and diseases of nerves as well as a break in continuity in the cerebro-spinal tracts, and which develops in that part of the neuron which is still in connection with the original cells (Monakow, Forel, Durante, Klippel, etc.). It refers to degenerative conditions which develop very slowly and irregularly, especially in young animals, and to diseases which have been present since childhood or which date back a long time. We class here atrophy of a nerve-nucleus after disease of the attached peripheral nerves, atrophy of the spinal roots and cord itself following upon an antedated amputation (Vulpian, Dejerine, etc.), the rarely observed ascending degeneration of the motor tracts, and the as yet obscure nuclear atrophies of the optic nerves and the primary optical centres after chronic lesions of the corresponding higher centres (V. Monakow, Moeli, etc.).

Through Nissl's and Marchi's method these observations have been much increased, and it appears that the basis of Waller's law will probably be shattered.

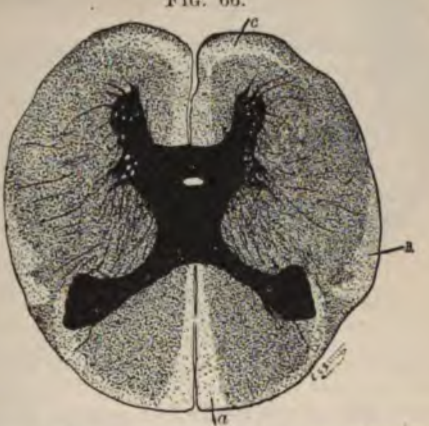
FIG. 65.



Section of the cervical cord in a case of amputation of the right arm. Retrograde degeneration.

We know now that soon (over twenty-four hours) after section of a peripheral nerve, even in the central stump and the nucleus belonging to it, certain alterations are detected that are only perceivable through the above methods (Marinesco, Nissl, Bergmann, Darkschewitsch, Flatau, etc.). The chief nucleus (*e.g.*, in section of spinal nerves the corresponding cell-group of the anterior horns) appears to degenerate first. Nissl's method shows splitting up of the granules and an eccentric displacement of the nucleus. (Fig. 67. Compare B with A.) A regeneration may soon occur if restitution takes place in the periphery. If this does not occur, atrophy may result in the cell.

FIG. 66.



Section of the upper cervical cord in a case of transverse lesion of the dorsal cord.

Marinesco distinguishes a stage of reaction (réaction à distance) which is shown by the above-mentioned chromatolysis and displacement of the nucleus, and a stage of regeneration in which the chief alteration is a swelling of the cell-body.

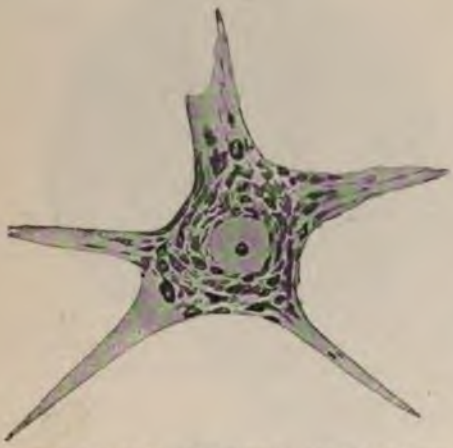
These conditions were first supposed to be a sort of retrograde degeneration which was in perfect antagonism with Waller's law. Later it was supposed that the ganglion cells of the nucleus belonging to it atrophy after the nerve-section, because the stimulations necessary to its existence—the sensory and central volitional impulses—do not reach it any longer (Marinesco-Goldscheider), and the transmission of excitation by the ganglion cells is hindered (Lenhossek). This is all, however, purely hypothetical. In harmony with the above, after section and other lesions of sensory nerves alterations of the cells of spinal ganglia have been found (Lugaro), and it has been shown that under certain conditions the "réaction à distance" is transmitted to the posterior roots and their prolongations, the posterior tracts (Redlich, Darkschewitsch).

Nissl's method appears to show, then, that a lesion of any part of a neuron may cause a degeneration of the whole neuron. But caution is necessary in accepting new conclusions, as the alterations do not seem to be permanent, and Nissl's bodies do not seem to be important elements of a cell.

Nissl's method has enabled us to study the diseases of the cells caused by poisoning, anemia, elevation of temperature, etc. Marinesco believes that these primary cellular changes are achromatophilous, and the above-mentioned secondary degenerations chromatophilous.

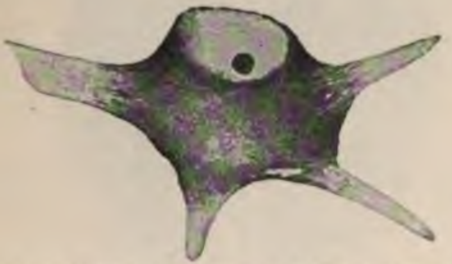
FIG. 67.

A.



Normal nerve-cell. Nissl's stain. (After Marinesco-Raymond.)

B.



Disease of the cell after section of the peripheral nerves. (Chromatolysis and nuclear displacement.)

SPINAL LOCALIZATION.

In spinal localization it is necessary to know accurately the different motor and sensory functions which each segment of the spinal cord governs.

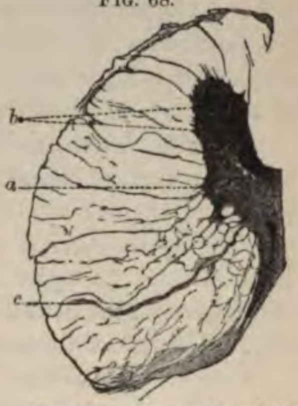
Our knowledge, not yet complete, is based for the greater part upon observations of Ross, Thorburn, Starr, Sherrington, Mills, Bruns, Kocher, Chipault, Démoulin, and myself.

It should be remembered not only that a nerve obtains its fibres from more than one root, but also that the motor fibres which pass from the anterior horn to the muscles are distributed, as a rule, over more than one root. The radicular fibres for a muscle arise, then, it seems, not from a single segment of the cord, but those adjacent have also a certain part in its formation. The destruction of the gray substance of one segment cannot therefore produce total atrophy of a muscle, as the adjoining segments will be able to nourish it to a certain extent. This is especially true of the *posterior* roots. Through anastomoses and the manner in which the roots spread out in the cord, the sensory nerves of a particular area of the skin are spread over two or three segments of the spinal cord. For this reason a lesion of one pair of posterior roots need not produce much disturbance of sensation; nor need a lesion at a certain height in the cord produce loss of feeling in the region innervated by the root arising from it.

The areas of innervation of single spinal roots are distinctly independent of the arrangement of the peripheral nerves. They overlap one another, each area being innervated by two or three sensory roots.

The nerves of the spinal cord, soon after both roots unite, divide into a ventral and a dorsal branch, the latter being the smaller, except in the two upper cervical nerves. The posterior branch innervates the muscles of the back, as well as the skin over the neck and back, but it has no part in the innervation of the extremities.

FIG. 68.



Part of a cross-section of the spinal cord of the upper cervical region. (After Grabower.) *a*, spinal accessorius nucleus; *b*, anterior nuclear groups; *c*, radicular fibres of the accessorius.

The motor fibres for the deep muscles of the neck and nucha arise in the first, second, and third cervical segments.

The levator anguli scapulæ, the trapezius, and the sternocleidomastoid are supplied by root fibres from these regions.

The nucleus of the spinal accessorius (Fig. 68) arises from a group of cells in the anterior horns of the upper cervical cord, and reaches downward nearly to the sixth cervical segment, though the three upper segments are the most important.

The platysma receives motor fibres from the third cervical segment (Kocher).

The phrenic nerve arises in the fourth cervical segment. It may also, perhaps, extend to the third. Probably fibres for the rhomboidei, supraspinatus, infraspinatus, and serratus anticus major (?) arise in this segment also.

The fifth and sixth cervical segment transmits through its anterior roots the motor fibres for the deltoid, biceps, brachialis anticus, and the supinator longus.

The fifth root is chiefly in play for these muscles; it appears also to innervate the supraspinatus and infraspinatus, the scaleni, the serratus anticus major, and the pectoralis major (probably only the clavicular portion).

The nuclear areas for the extensor of the hand and the extensor communis digitorum are found in the sixth and especially the seventh cervical segments. The sixth also sends fibres to the pronators and the triceps.

The fibres for the long extensor of the hand and the fingers arise in the seventh cervical segment. This also contains the innervation centres for the latissimus dorsi, teres major, triceps, and perhaps also the flexor of the hand. The extensor nuclei, however, lie above those for the flexors, and the supinator nuclei probably above those for the pronators.

The flexors of the fingers are mostly innervated from the eighth cervical segment, also the small muscles of the hand and the extensor pollicis brevis and longus.

The first dorsal segment takes part in the innervation of the small muscles of the hand, and here is also the origin of the oculo-pupillary fibres of the sympathetic. (Dastre and Morat believe that the eighth cervical and first two dorsal roots contain vasodilatory fibres for the face.)

Innervation of the Skin.—The second, third, and fourth posterior cervical roots innervate the skin of the head, nucha, neck, and upper part of the breast, as far as the second intercostal space, with sensory fibres.

The fifth innervates the shoulders over the deltoid downward from the joint, also the lateral areas of the upper and lower arm to the styloid process of the radius.

The eighth cervical and the first dorsal innervate the skin of the inner side of the upper and lower arm and the ulnar area of the hand and fingers. It is denied by some that its branches spread over the upper arm. The rest of the arm is supplied by the sixth and seventh cervical.

Kocher believes that the sixth cervical alone innervates the radial and median areas of the hand.

The motor branches of the second to the seventh dorsal nerves innervate the corresponding intercostal muscles, the levatores costarum, the triangularis sterni, and the upper part of the rectus abdominis; the eighth to the twelfth, all the abdominal muscles, excluding the intercostal muscles. The dorsal cord innervates with motor fibres the back also, with the exception of the shoulder, which is supplied by the cervical.

The sensory innervation of the dorsal nerves is confined to the region extending from the second intercostal to the symphysis, and behind to the gluteal region. It extends downward, therefore, much farther than the course of its chief branches. This shows, as Sherrington first observed, that every nerve innervates an area which extends upward and downward into the region of the neighboring nerves. The sensory areas on the skin corresponding to the dorsal segments do not run parallel to the ribs and intercostal nerves, but horizontally forward and backward. Therefore the upper border of a sensory area lies three or four spinal processes further down than the exit of its nerve from the spinal canal.

Concerning the innervation areas of the lumbar and sacral roots our knowledge is not so accurate.

FIG. 69.

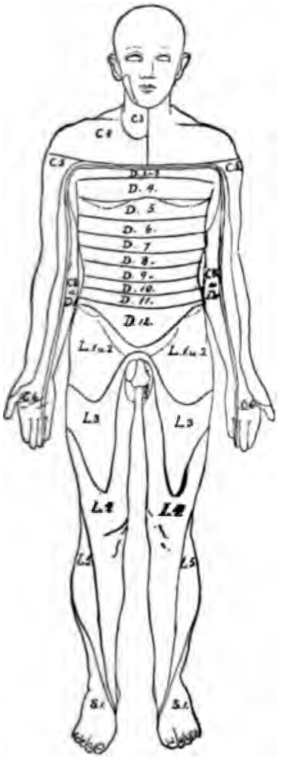
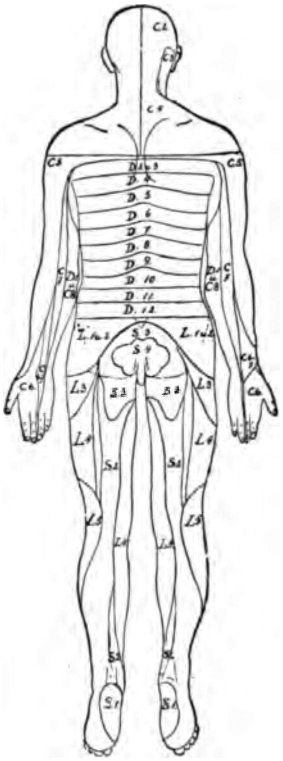


FIG. 70.



Showing the regions innervated by the different spinal roots or the corresponding segment of the cord. It should be remembered that the limits are not in reality so sharply defined, but extend into one another. (After Kocher, with some modifications from other authors.)

The first lumbar segment partly innervates the abdominal muscles and also the iliopsoas.

The second and third contain the trophic centres for the cremaster and the flexors and the adductors of the upper thigh, probably also for the sartorius.

The third and fourth contain centres for the extensors and adductors of the thigh, the abductors (?), and the extensor cruris quadriceps, probably also the tibialis anticus.

The fifth lumbar and the first sacral, for the flexors of the knee, the gluteal muscles (also, probably, the pyriformis, obturator internus, and gemelli), and the long extensor of the foot and toes; which, however, is also innervated by the first sacral.

The first and second sacral segments give origin also to the fibres for the muscles of the calf of the leg and for the small muscles of the foot.

The perineal muscles, the bladder, and the rectum are innervated by the third and fourth.

The fifth sacral and the coccygeal innervate the levator ani.

Figures 69 and 70 will convey much knowledge concerning the innervation of the skin. It must be remembered, however, that the limits are not in reality so sharply defined, but that the areas overlap one another so that every region is innervated by more than one root, and that individual variations occur.

The third and fourth sacral roots contain the sensory fibres for the perineal region, the anus and its surroundings, and the mucous membrane of the bladder, as well as for a small strip of skin on the posterior and inner surfaces of the thigh. Sherrington calls this part of the skin the *genital flap*, or sexual skin.

The conus medullaris contains the motor centres for the bladder and anus in its lower part, at about the place of origin of the fourth sacral nerve, while the corresponding sensory area lies higher (?). The erection reflex is supposed to be in the second sacral segment, while the ejaculation centre lies deeper. (Kocher, Schlesinger.) In diseases of the conus, notwithstanding anesthesia of the scrotal skin, the scrotum is very sensitive, the cremasteric reflex being also intact.

The sexual desire and the ability for erection may be intact, notwithstanding the failure to ejaculate semen.

The abdominal reflex comes from the eighth to the twelfth dorsal roots, the cremasteric reflex through the first and second lumbar roots. The reflex-arc for the knee-jerk is in the second, third, and fourth lumbar segments and their roots. The Achilles tendon reflex is innervated from the fifth lumbar and first sacral roots; the plantar reflex from the first and second sacral roots. These data are not absolutely and positively proved, however.

It has been attempted (Kaiser, Collins, Hammond) to ascertain the position and extent of the different spinal nuclei by anatomical examinations. These examinations appear to show that the median nucleus of the anterior gray matter of the dorsal cord innervates the muscles of the back. A phrenic nucleus and a nucleus for the quadratus femoris have also been separated, though these results are too uncertain to deserve further consideration here. Sano has lately made special investigations in this direction, using Nissl's method. His results also have not been confirmed.

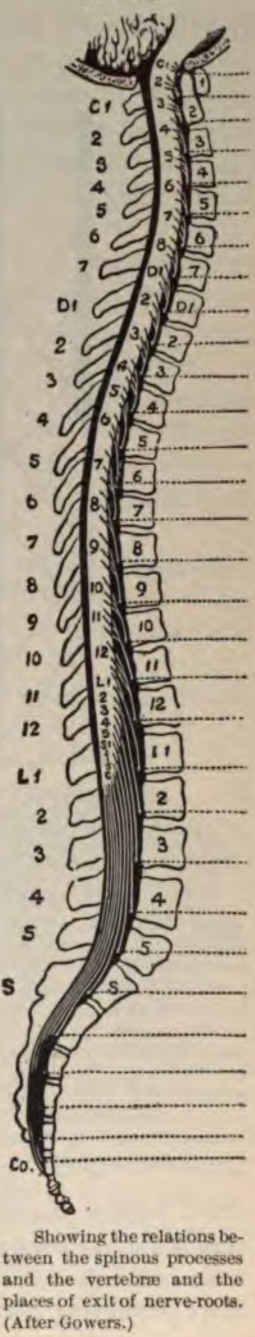
An atrophic paralysis of any muscular region, if spinal in origin, permits us, then, to form definite conclusions as to the location of the trouble. It is necessary to be very cautious in forming conclusions from disorders of sensibility. Complete anesthesia ensues only when the neighboring roots and segments are involved.

A lesion of the sixth dorsal segment, for example, produces anesthesia of the skin innervated by it only when the fifth and probably the fourth are also involved. As we find that in partially impaired root zones hyperesthesia often develops, we can locate the disorder, as this always indicates the upper border of the process. The reflexes also indicate much in this direction. It should not be forgotten that diseases of the spinal cord which produce a complete break in continuity may cause, even when their seat is above the reflex centres, an annulment of the reflexes, especially the deep reflexes. (Bastian, Bruns, etc.)

The relation of the segments of the spinal cord to the vertebræ and of the origin of the roots to their place of exit is of importance.

The origin in the cord lies higher than the place of exit in the canal,—with the exception of the upper cervical part,—and this condition increases as we descend. Individual differences are, however, very marked. (Reid, Starr.) As a rule, the seventh cervical vertebra corresponds to the first dorsal segment, the eighth cervical root arising opposite the lower border of the sixth cervical vertebra. In the dorsal region the roots arise in the cord one or one and one-half to three vertebræ above the place at which they emerge from the canal. The difference increases from above downward. The sixth dorsal root arises at a point corresponding to the fourth and fifth vertebræ, the tenth root at the eighth vertebra. The first lumbar nerve arises opposite the eleventh dorsal vertebra; the second between the eleventh and twelfth; the third and fourth op-

FIG. 71.

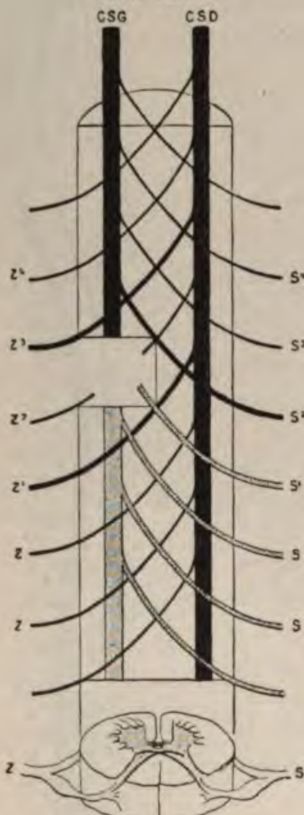


Showing the relations between the spinous processes and the vertebræ and the places of exit of nerve-roots. (After Gowers.)

posite the twelfth; the fifth lumbar and first sacral between the twelfth dorsal and first lumbar; the other sacral nerves opposite the first lumbar vertebra and the interarticular cartilage between the first and second. The whole lumbar and sacral cord corresponds to the part of the spinal canal between the eleventh dorsal and first lumbar inclusive.

The nerves from their origin to their place of exit descend more or less along the cord, and through this the roots of a segment override a greater or lesser number of the roots arising below it. This is especially true of the lumbar roots.

FIG. 72.



Showing the sensory relationship in Brown-Séquard's paralysis. CSG and CSD, sensory conducting-tracts of the right and left side; S, S^4, posterior roots of the left side; Z, Z^4, posterior roots of the right side. The incision between Z^2 and Z^3 represents the unilateral lesion. The anesthesia affects the roots of the left side (S, S, S) entering the cord below the lesion, the root Z^2 of the right side which enters the lesion directly, and hyperesthesia develops in the region innervated by Z^1 and Z^3 of the right and S^2 of the left side. (After Brissaud.)

UNILATERAL LESIONS OF THE SPINAL CORD; BROWN-SÉQUARD'S PARALYSIS.

Brown-Séquard showed experimentally what symptoms a unilateral lesion of the cord produces. They are as follows:

Paralysis on the same side, anesthesia upon the opposite side. The anesthesia is an incomplete one, as the muscular sense is left intact, though decreased or lost on the paralyzed side. We find on the paralyzed side a hyperesthesia especially to pain, and also generally an increase of temperature of about 0.5° to 1.0° C. The bladder and rectal functions may be disturbed, but this is no constant sign.

The reasons for such symptoms are supposed to be the following: Paralysis of the same side is produced because the direct pyramidal tract which is chiefly in question here is a direct tract in the spinal cord. The sensory tracts are supposed to decussate immediately after their entrance into the spinal cord, the fibres conducting the muscular sense being the only ones not doing so.

The researches of Mott and Turner only partly confirm the results of Brown-Séquard.

Clinical observations, however, agree with Brown-Séquard's teachings, and recent views seem to be in harmony with them.

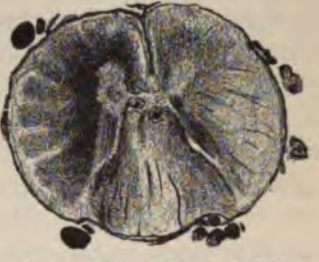
The dorsal cord is most often affected in this manner. We find, then, (*a*) upon the *same* side: 1. Paralysis of the legs, with increased knee-jerks, though these may at the start be weakened or annulled (and, as I once observed, for many weeks). 2. Hyperesthesia of the skin, for pain especially. 3. Loss of muscular sense. (The condition of this sense is variable, but is generally lost on the paralyzed side.) 4. With the return of motility ataxia may set in in the legs (Bottazzi, Herhold, Oppenheim). (*b*) On the *opposite* side: Anesthesia to all conditions except the muscular sense, or, more commonly, decrease of pain and temperature senses.

There exists upon the side corresponding to the lesion in the same root zone a semi-girdle zone of pain and hyperesthesia or anesthesia, while the anesthetic zone upon the opposite side is bordered above by a hyperesthetic zone. Fig. 72, after Brissaud, attempts to show this; it indicates only sensory fibres.

If the disease is in the cervical cord, we find spinal hemiplegia,—*i.e.*, paralysis of the leg and arm of the same side and anesthesia of the trunk and leg of the other side. Disturbances of the pupils may also take place on the side on which the lesion occurs.

When a unilateral lesion attacks the lower lumbar or sacral cord, the paralysis and in general the sensory disturbances are on the same side, as here very few sensory roots cross to the other side.

FIG. 73.



Cross-section of the spinal cord from a case in which a Brown-Séquard's paralysis had been formerly present. (Weigert's stain.)

It is thought to-day, from recent studies of the course of the fibres in the cord, that the muscular sense is transmitted without decussation by the posterior tracts, probably in part by Clarke's columns, through the direct cerebellar tracts. The tracts for pain and temperature take their course through the gray matter. It is improbable, however, that they ascend in this. They mostly, or perhaps altogether, reach the antero-lateral tract of the opposite side,[1] though it is not known whether this decussation occurs in the anterior or the posterior commissure. Though the gray matter in general merely transmits the sensory fibres as they enter the cord at the affected area, the antero-lateral column of the dorsal cord contains all the conducting paths for pain and temperature sense of the legs of the opposite side.

---

[1] Woroschiloff, Holzinger, and Bechterew found that section of the lateral tract produced analgesia. It has been stated that the lateral tracts contain paths for the sense of touch which come directly from the posterior roots and ascend without decussation. This has, however, not been proved.

The conducting-paths for tactile sensation, for the most part, seem to pass up the posterior tracts without decussation. This should produce in unilateral lesions tactile anesthesia of the same side. Clinically, however, this is not the case. A satisfactory reason for the hyperesthesia of the paralyzed side is not known, notwithstanding the studies of Enderlen, Raymond, etc.

Pathology teaches us that it is especially in cases of stab wounds of the cord that Brown-Séquard's symptoms are found. Hemorrhages also may cause them, as well as tumors, spinal lues, rarely myelitis and sclerosis. The symptom-complex generally is incomplete, the paralysis being more marked in one leg and the anesthesia in the other. It is the rule that signs of a diffuse lesion (paraplegia or parparesis) precede the development of this symptom-complex, especially when due to traumatisms.

The prognosis of unilateral lesions of traumatic origin is good, inasmuch as a partial if not a complete retrogression of symptoms occurs.

THE ARTERIES OF THE SPINAL CORD.

The arteries which nourish the cord arise from the spinal, intercostal, lumbar, and sacral arteries. The spinal arteries come from the vertebral, there being two anterior ones, which generally unite, and two posterior ones. The spinal arteries run along the anterior and posterior surfaces of the cord and everywhere give off small branches. The branches from the intercostal, lumbar, and sacral arteries run alongside the spinal roots and communicate with the vessels from the spinal arteries. This produces the so-called arterial chains, the tractus arteriosi, an anterior and two lateral ones. The branches pass partly from the floor of the anterior fissure to the central part of the cord—these supply the gray matter, with the exception of a large part of the posterior horns—and partly from the periphery to the white matter and the posterior horns. The anterior tract gives off branches at all heights, which pass as *arteriæ sulci* into the spinal cord and reach to the anterior commissure. Here it divides (Adamkiewicz) or bends undivided (Kadyi) right or left as the *arteria sulco-commissuralis*, enters the anterior horn, and supplies the greater part of this and a small area of the surrounding white matter. One branch reaches Clarke's columns.

In addition to these central arteries we have the *vasocorona*, which enter the cord from the periphery. Although the white matter and the gray matter are not supplied by two entirely independent arterial systems, there are areas which are supplied only by the central arteries and others in which we find only the vasocorona.

CLASSIFICATION OF THE DISEASES OF THE SPINAL CORD.

The diseases of the spinal cord are confined to single tracts or systems of fibres, or to sections of definite physiological functions (system diseases), or they spread diffusely over the organ (diffuse diseases). There are also diseases which are supposed to be spinal in their nature, but which have no known pathologico-anatomical basis.

THE SYSTEM DISEASES OF THE SPINAL CORD.

TABES DORSALIS, LOCOMOTOR ATAXIA.

Tabes dorsalis is the most common of all spinal diseases, being especially frequent in the large cities. It is found in men more often than in women, and especially in those of middle age. It has been reported as occurring in childhood,[1] but no true tabetic lesions have been noticed in children's cords on pathological examination. It is rarely observed in the aged.

Etiology.—That there is a connection between *tabes* and *syphilis* has been shown by statistics (Erb, Fournier). Persons with no history of syphilis rarely become tabetic. Tabes is, however, not a syphilitic disease in a pathologico-anatomical sense. The pathological alterations have nothing in common with those of visceral syphilis. It is possible that through a syphilitic taint, poisons are formed in the body which produce a simple degeneration of certain parts. Probably other poisons may also induce the disease. Tuczek has shown that *ergotin* produces a disease which in symptomatology and anatomical lesions is similar to tabes, but which, unlike tabes, is not progressive. Certain cases seem to show a relationship between tabes and chronic lead-intoxication.

Tabes generally results from five to fifteen years after the syphilitic infection; rarely earlier or later. The syphilitic symptoms are generally slight; often it is possible only to ascertain that a venereal ulcer had been present, without being able to determine its character.

Hitzig is of the opinion that the virus which causes tabes may also come from a soft chancre.

---

[1] Cases of this kind have been described by B. Remak, Kellog, Gombault and Mallet, Siemerling, Bloch, Raymond, etc.; most of them were cases of cerebro-spinal syphilis, though Raymond and some other observers claim to have seen real infantile and juvenile tabes.

I know of two patients in whom the disease occurred in middle age, though neither case gave a history of syphilitic infection, whose fathers, however (one was a tabetic), had had syphilis. Erb and Fournier have noticed the same. Tabetic individuals rarely show any syphilitic symptoms. In women even a history of infection is often wanting. We do not know any other cause. Hereditary influence may predispose towards it, and exposure to cold, over-exertion, traumata, sexual excesses, alcoholism, etc., may assist as causes.

Symptomatology.—There are varied and manifold phenomena and symptom-complexes, though there are a number of symptoms which occur in almost all cases. In the early stages these often form the only signs, and in later ones they are the surest indications for diagnosis.

The first stage is characterized particularly by the following symptoms:

(1) Westphal's sign,—*i.e.*, absent knee reflexes,—(2) Argyll-Robertson pupil, (3) lancinating pains, (4) analgesia.

Westphal's sign may occur years before all other symptoms, and is rarely absent at the beginning. It may be present in only one knee.

The *Argyll-Robertson pupil* is the next important symptom. It may be the only indication of future tabes, but generally is not so constant a symptom as that of Westphal. It may be present only in one eye, and is often preceded by a sluggish or weakly-reacting pupil. The contraction of the pupil resulting from convergence generally remains normal, notwithstanding existing pupillary rigidity, though there are exceptions.

The *lancinating pain* is a subjective symptom, and hence diagnostically is not as important as the two preceding ones. The pains are very severe, are paroxysmal, and are lightning-like in character. The intervals without pains may last days, weeks, or months. The attacks may be so short that only upon inquiry does the patient remember about them, and may persist for days or weeks, but generally last only a few hours. They occur especially in the lower extremity, but may manifest themselves anywhere, may extend over a large surface or only for a short distance, and may take any direction. Occasionally the pain invades a circumscribed area of the skin, and seems to bore its way in from there. The skin on this spot may be so hyperesthetic that even the bedclothes cause pain when they touch it. In later stages we may also often find dull, long-drawn-out, boring pains. We often find a girdle pain; sometimes a painful sensation in the anus, as if a stick had been forced into it. These lancinating pains are a regular symptom, are rarely absent, and may precede the graver symptoms by from ten to twenty-five years.

The *analgesia* is generally found in the lower extremities. While the general sensibility is unimpaired, we find that prickings with a needle produce no pain, or but very little. If we lift a part of the skin and pierce it with a needle, the patient feels that he has been touched, but has no pain.

We may, perhaps, add the *swaying with closed eyes* (*Romberg's symptom*) as a symptom of the first stage, though it does not generally occur so early in the course of the disease as the above four symptoms. Let the patient place his feet together and stand upright; at times a swaying occurs, which becomes more marked when his eyes are closed. A slight sway occurs in healthy individuals, especially if they are frightened. The pathological condition is made more conspicuous if the patient is compelled while his eyes are shut to bend his back and then straighten himself again. (Oppenheim.)

Other symptoms which may occur in the early stage are difficulty of urination, sexual weakness, and often paralysis of the eye-muscles. Gastric disorders, spasmodic cough, paralysis of the vocal cords, diseases of the joints and bones, may all be present at this stage.

The disease reaches its full development when ataxia occurs. The disorder of movement, at least as far as it is found in the extremities, is not motor weakness, but *incoördination*. The individual movements of the arms and legs are executed with full force and amplitude, not in the shortest possible manner, but in jerks and with the dissipation of much muscular energy.[1] This incoördination attacks first the muscles of the lower extremities, the patient noticing uncertainty in walking (especially in the dark) and in descending stairs. It often is objectively noticed before the patient is aware of it. The nature of ataxia and the manner of examination for it have already been described. It is noteworthy that it is especially prominent when the aid of the eyes is absent. A tabetic lessens his coördination by using his eyes as a control.

The ataxia of the legs produces a characteristic gait. When fully developed, it is of the following nature. The patient lifts the leg very high by excessive flexure and outward rotation of the hip-joint, and forcibly throws it down by excessive extension of the leg and foot, so that the foot comes down with a stamp, the heel touching the floor first; he walks rapidly, with unequal strides, eyes constantly on the floor, and is in danger of falling the moment he looks away. Sometimes his knees suddenly give way and he falls.

The ataxia may be observable in a recumbent position and not noticeable in walking, or *vice versa*. Slight degrees are detected by the heel coming down with a stamp.

---

[1] Most investigators regard the ataxia of tabes as being a result of sensory disturbance, particularly the impairment of deep sensibility. Others believe it to be due to the loss of excitations which normally pass from the periphery to the spinal cord and influence the motor functions unconsciously.

The upper extremity may be the first attacked. The finer, complicated movements of the hands are executed with difficulty, as buttoning of the clothes, writing, etc.

A peculiar phenomenon often occurs in connection with the ataxia, especially in the arms. The patient cannot keep them quiet, even if they are supported. He moves his fingers involuntarily, or the hand may be lifted or the whole arm may be elevated to a vertical position without the patient being aware of it (*spontaneous movements*).

The facial and glossal muscles are rarely involved. Ataxia is not an early symptom of tabes; it is developed only after many other symptoms have appeared. There occurs at the same time an atony of the muscular system which causes an abnormal freedom of passive movements.

The symptoms which follow may occur at any stage of the disease. The most common and important will be first discussed.

We have already spoken of the pains. *Paresthesias* of the most varied types form an almost constant symptom: a feeling of formication, numbness, etc., especially a sensation as if the patient were walking on rubber or felt, or as if the pelvis were bound by a rope or a hoop. He often complains of an intense coldness, particularly in his legs. The paresthesia generally is not found in a certain nerve-zone in the lower extremities; in the arms, however, it generally attacks the skin supplied by the ulnar nerve first, especially the fourth and fifth fingers.

The analgesia already spoken of seems to be the earliest objective symptom of disordered sensibility. More rarely a hyperesthesia occurs first, so that the slightest touch—especially in the gluteal region—produces marked pain. In the later stages, rarely in the first, we find decreased tactile sensation as well as decrease of the muscular and pressure senses. The tactile anesthesia, according to Hitzig and Lähr, first attacks the gluteal region, the region of the middle dorsal nerves. The stereogenetic sense is rarely involved, except in the last stage.

The sensory disturbances may involve one or both legs or other regions at the same time; but generally only small circumscribed areas are attacked. We often find delayed sensation in which pain may be detected only after from two to five seconds. Naunyn and Remak have described a so-called double impression, in which a needle-prick at first merely produces a feeling of touch, and only after a certain interval is pain noticed. Once I observed that a single needle-prick produced a feeling as if the skin had been pricked in two places. Occasionally we find that at the place where mere pricking of the skin does not produce pain we may evoke a severe and prolonged pain by drawing the pin-point along the skin.

Deep sensibility may be impaired in the initial stage, but this is generally not the case, it coming on in most cases later in the disease. In advanced cases the patient may have no idea of the position of his limbs, whether they are in the bed or outside of it.

The bladder is almost always affected. The first indication is generally a difficulty in urination. The patient may also tell you that he can retain his urine very long, and that he experiences a desire to urinate only once or twice a day. This is an evidence of a pathological condition. Incontinence and retention, however, are later symptoms, and, notwithstanding the progressive nature of the disease, may in time disappear.

Obstipation rarely occurs. *Impotence* may be an early symptom; so may *satyriasis*. In one of my cases I found an involuntary priapism that continued for weeks at a time, and which induced painful urination; this was succeeded by impotence. Nightly attacks of priapism with incontinence of the urine occur. We call those paroxysms of lustful feeling with vulvovaginal secretion which are found in tabetic women *clitoric crises*.

The most frequent disease of the cranial nerves present in tabes is *paralysis of the ocular muscles*. It may affect any of the muscles, and is generally *transient*,—*i.e.*, may disappear and reappear. A chronic paralysis occurs only in the late stages. It is never associative, but may occur in any of the muscles of one or both eyes; *ophthalmoplegia* is noticed sometimes. Often, even very early in the course of the disease, we find a myosis; more rarely, a mydriasis.

The optic nerve is often involved; it furnishes a typical picture. We find a *simple, progressive atrophy of both nerves*, which commences early and leads to complete blindness. Though one eye may be involved more than the other, and earlier, it is almost never limited to the one eye during the whole course of the disease. The optic atrophy is as a rule an early symptom. It may be for a long time the only tabetic symptom.

The trigeminal area may also be involved and show symptoms of sensory disturbance of every nature.

Trophic disturbances, as neuroparalytic keratitis, ulceration of the mucous membrane of the mouth, and facial hemiatrophy, herpes, etc., are rarely observed. Sometimes the teeth fall out spontaneously without pain or bleeding. We sometimes, though rarely, find that the sense of taste has been lost. One of my patients complained that he tasted his food only at the first bite, and that afterwards he did not taste anything; another had no sense of taste except during the act of deglutition.

Only rarely do we find weakness or atrophy of the muscles of mastication (Schultze). The senses of smell and hearing are rarely involved.

Very important are the symptoms which arise from involvement of the vago-accessorius. We include here the rapid pulse and the heart crises, a cardiac pain accompanied by a feeling of oppression, and a rapid and irregular heart. This condition is very similar to that of *angina pectoris*.

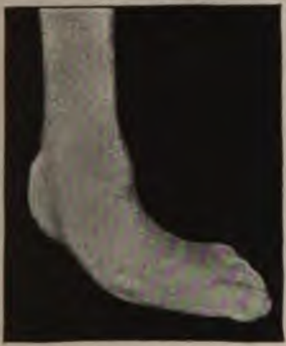
Gastric crises (Topinard, Delamarre), or convulsive vomiting, occur more commonly. The attack commences suddenly, the patient experiences a marked pain in the gastric region or a feeling as if his stomach were being twisted into a knot. He commences to vomit,—first his food, next gall. The vomiting and pain continue, notwithstanding the complete withdrawal of food. The patient loses strength, his face becomes pale and sunken, the urine scanty and concentrated, the pulse rapid and irregular. The tongue is generally normal. Hours, days, or weeks after the onset it suddenly ceases, and the patient may again eat. The intervals between the attacks are variable. We may find in some cases pain without vomiting, or vomiting without pain. I had a case in which glycosuria developed, and later all the symptoms of diabetes mellitus, which, however, eventually disappeared. *Laryngeal crises*, or spastic cough, are more rare (Féréol). There is a sudden feeling of choking, with a labored inspiratory stridor of some seconds' or minutes' duration, or a paroxysm similar to that of whooping-cough, in which the cyanosis and want of breath become considerable. In one of my cases spasms of sneezing were combined with these attacks of cough. A laryngoscopic examination is always negative. Lately, however, many observations have shown the presence of paralysis of the laryngeal muscles in tabes. The most common is a paralysis of the crico-arytenoidei postici, which separate the vocal cords. I found in these cases a complete loss of reaction of the recurrent laryngeal to electrical excitation. At times we may discover a point on the neck between the larynx and the sternocleidomastoid, pressure on which produces pain. Attacks of coughing have been produced by pressure upon this point.

Pharyngeal crises (Oppenheim) are rare. In such attacks movements of deglutition follow each other rapidly, a gushing murmur being heard at the same time. The attack lasts from some minutes to a half-hour; twenty-four acts of deglutition may occur in a minute. We characterize as *intestinal crises* attacks of colic with diarrhœa; as *kidney and bladder crises* attacks of pain in these regions, etc. A sort of globus is sometimes observed.

Trophic Disturbances.—Trophic disturbances of the joints and bones occur very frequently. The *tabetic arthropathies* appear generally in the early stages, the knee-joint being most commonly affected. They occur suddenly; the joint swells and edema supervenes. The accumulation of fluid may be considerable.[1]

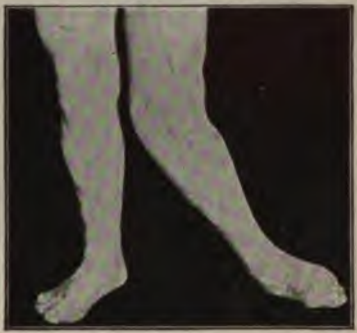
No pain, reddening, or fever occurs. The joint is rapidly destroyed, and this, with the loss of contraction of the capsule, produces a condition similar to a luxation or a subluxation (Figs. 74 to 77). At the same time new bone is rapidly formed, and the formation of excrescences and free bodies increases the volume of the joint. This course is similar to that of arthritis, except that its manner of development and the absence of pain differentiate it from the latter. The hip-joint may also become involved (Fig. 79). The shoulder-joint (Fig. 78) and elbow-joint, the ribs, and the ankle-joint are rarely attacked. Arthropathy of the foot may produce the curious deformity called (by Charcot and Féré) *pied tabétique* (Fig. 74). I observed a spontaneous fracture of the thigh in two of my cases. Arthropathy and fracture often occur together (Rotter).

FIG. 74.



Tabetic foot.

FIG. 75.



Genu inversum in tabetic arthropathy.

Other trophic disorders are,—

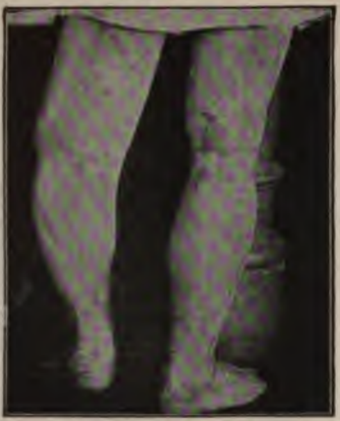
1. *Malum perforans*, or perforating ulcer,—a round ulcer, developing generally on the plantar surface of the foot, especially on the ball of the great toe, which usually bores inward, and which resists most therapeutic measures. There is also a milder form. 2. *Spontaneous gangrene* (Joffroy, Pitres) has been observed in isolated cases. Many nutritional disturbances of the skin and nails, including the spontaneous falling out of the latter, have been described.

Hemihyperidrosis unilateralis, suggillation, herpes, and other exanthems may occur.

---

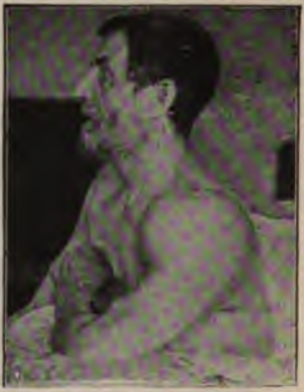
[1] I have also noticed and described accumulations of blood in the joints. The French writers (Brissaud, Charcot-Dufour) described this phenomenon afterwards under the name "Hémarthrose tabétique."

FIG. 76.



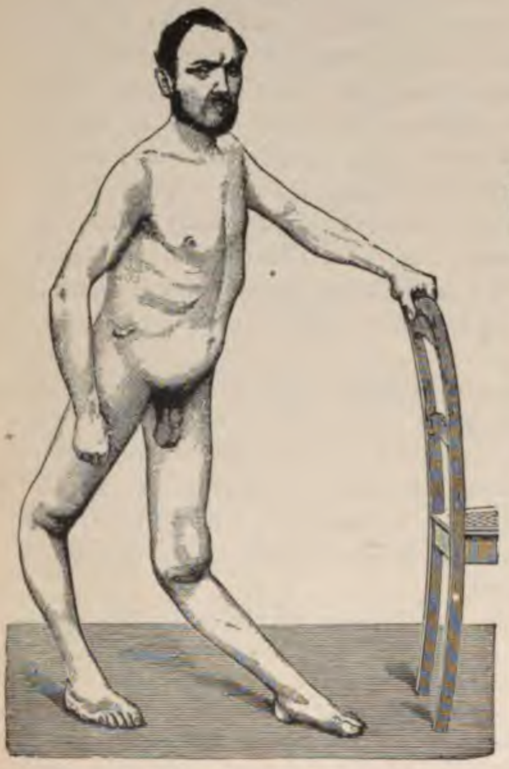
Genu inversum in tabetic arthropathy of the left knee-joint. (Swelling in the neighboring tissues also.)

FIG. 78.



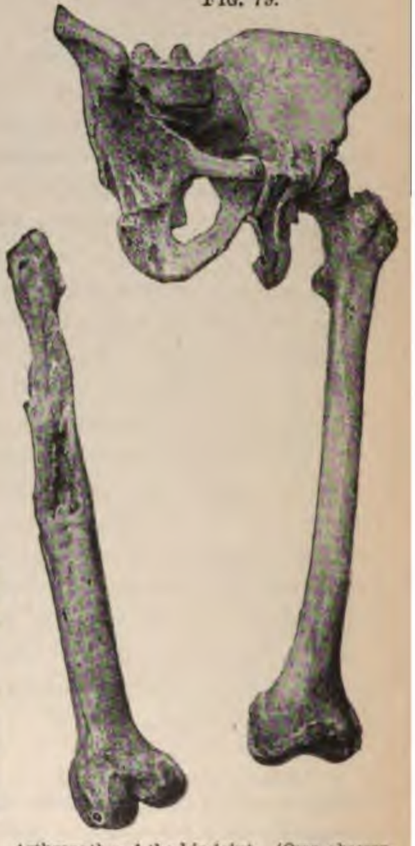
Tabetic arthropathy of the left shoulder-joint.

FIG. 77.



Deformity resulting from tabetic arthropathy of the knee-joint. (After Westphal.)

FIG. 79.



Arthropathy of the hip-joint. (Own observation and after Sonnenberg.)

The apoplectic attacks which may occur in tabetics are probably not a symptom, but result from lesions, probably syphilitic, in the cerebrum. *Insufficiency of the aortic valves* is also only a complication of tabes.

Diagnostic Remarks.—Advanced cases are not difficult to diagnose. It may be confused with multiple neuritis. This disease, however, is an acute or subacute one, reaches its height after a few weeks, or, at the most, a few months, to end in death or a slow convalescence. Multiple neuritis may have the pains, sensory disturbances, ataxia, Westphal's and Romberg's symptoms, in common with tabes; but the bladder disturbance, girdling sensation, and Argyll-Robertson pupil are wanting. Progressive bilateral atrophy of the optic nerves does not occur in polyneuritis, though we may find an optic neuritis leading to partial atrophy or to a central scotoma (especially in alcoholic cases). On the other hand, we find in polyneuritis pain on pressure upon the peripheral nerves and muscles, as well as a degenerative paralysis of the peripheral nerves. Psychical disorders also may occur in multiple neuritis unlike any found in tabes. Moreover, the etiology shows syphilis as a causative factor in tabes; an intoxication (alcohol, lead, arsenic, etc.), or an infection (acute infectious diseases, tuberculosis) as a cause of multiple neuritis.

A form of neuritis which, though it has much in common with tabes, has a very different prognosis is *diphtheritic paralysis.* In the generalized form, which is not so rare, we find ataxia, Westphal's symptom, sensory disturbance of the limbs, Romberg's sign, eye-muscle paralyses, etc. The history of the case, however, always shows us the nature of the trouble. We learn that a few weeks or months before there occurred a throat-trouble, accompanied by fever, that difficulty in deglutition, nasal voice, pharyngeal and laryngeal paralysis, etc., supervened, and that only after these did the first-mentioned symptoms occur.

Compare also the remarks concerning "acute ataxia" in the article on myelitis. Tabes dorsalis and diabetes mellitus have also many things in common.

Glycosuria may be a symptom of tabes; the pains, Westphal's sign (Bouchard), disorders of sensation, paralysis of eye-muscles, the spontaneous falling out of the teeth, the impotence, the attacks of vomiting, etc., may all occur in diabetes.

The basis for such symptoms is probably a neuritis, so that such a symptom-complex is a diabetic pseudotabes. Whether bladder paralysis and Argyll-Robertson pupil may occur from this cause is doubtful. In some cases of diabetes a degeneration of the posterior cords has been observed (Williamson, Souques, Marinesco). It may also happen that both diseases occur together.

Addison's disease may present some tabetic symptoms, as Westphal's symptom. But as soon as the pigmentation is marked, no error is possible. Bonardi has observed a degenerative process in the posterior cords in this disease.

In general, only in the initial stage can the diagnosis be difficult. It may present many symptom-complexes at this time,—

1. Lancinating pains, Westphal's sign, Argyll-Robertson pupil.
2. Weakness of the bladder, Westphal's sign, girdle-sensation.
3. Optic atrophy, Westphal's sign, and a tabetic cuirass (girdle-sensation), with corresponding hyperesthesia and analgesia.
4. Impotency, lancinating pains, optic atrophy.
5. Attacks of vomiting, Westphal's sign, or Argyll-Robertson pupil.
6. Diseases of the joints, analgesia, Westphal's sign, or Argyll-Robertson pupil.
7. Paralysis of the vocal cords (with or without attacks of coughing), Westphal's sign, Argyll-Robertson pupil.
8. Spontaneous falling out of the teeth, with disturbance in the sensory area of the trigeminal, Westphal's sign, bladder-trouble, etc.
9. Paralysis of ocular muscles, girdle-sensation, analgesia, etc.

Further combinations need not be given, as it can be seen from the above what manifold differences may exist in cases of early tabes.

When we find only those symptoms which occur also in early dementia paralytica, a diagnosis is difficult. Argyll-Robertson pupil or Westphal's symptom occurring alone will not make a diagnosis of tabes. It is different, however, with the simple progressive optic atrophy; this alone will be sufficient to make at least a probable diagnosis of incipient tabes.

The optic atrophy, the Argyll-Robertson pupil, and Westphal's sign are not rarely forebodings of dementia paralytica. We must then look for psychic disorder, speech disturbances, or paralytic attacks to make a differential diagnosis. Guard, however, against thinking of dementia paralytica simply because psychical alterations occur in the course of a tabes.

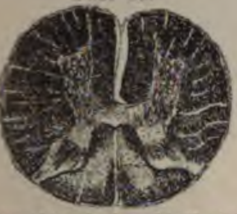
V. Strümpell speaks of a chronic nicotine intoxication similar to tabes, but does not describe it. See the chapter on syphilis of the spinal cord for a differential diagnosis between tabes and syphilitic pseudotabes.

Pathological Anatomy.—The chief pathologico-anatomical alteration is the *gray degeneration of the posterior tracts.* This may even be seen macroscopically: the posterior tracts appear gray and smaller and more shrunken than is normal. Only in cases which have reached an autopsy early can it not be detected with the naked eye.

A microscopical examination reveals that the process begins in Burdach's column, and generally in the upper lumbar segment. First, two symmetrical fields degenerate (Figs. 80 and 81), which are in the area of the initial zone of the posterior roots,—*i.e.*, that part of the posterior tract in which the posterior roots are formed. The degeneration of Goll's tracts above is in part a direct result of this atrophy of Burdach's columns.

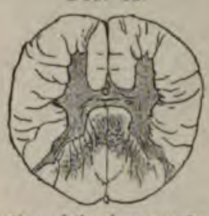
In advanced cases the whole posterior tract in the lumbar and dorsal cord is degenerated (with the exception of a small area alongside the posterior commissure). In the cervical cord the disease is confined at first to Goll's tracts; later the outer parts of the posterior cords degenerate. The degeneration then appears as in Fig. 82.

FIG. 80.



Cross-section of a spinal cord in the initial stage of tabes dorsalis. (Weigert's stain.)

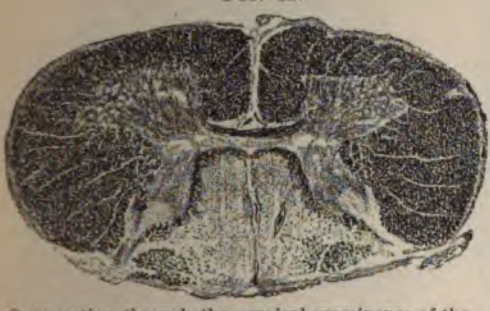
FIG. 81.



Localization of the degeneration of the posterior cord. Diseased part shaded. (After Westphal.)

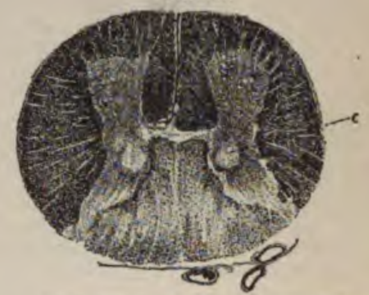
The axis-cylinders degenerate; the glia remains normal or proliferates.

FIG. 82.



Cross-section through the cervical prominence of the spinal cord in advanced tabes. (Weigert's stain.)

FIG. 83.

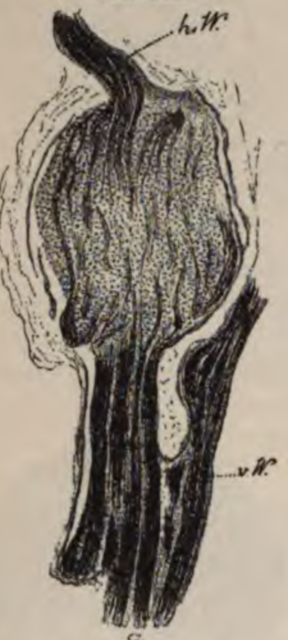


Degeneration of the posterior tracts and Clarke's column in tabes dorsalis. *C*, Clarke's column. (Weigert's stain.)

The disease does not, however, confine itself to the white matter: the fibres of the gray, particularly (1) Clarke's column (Fig. 84) and (2) the posterior horns, and especially Lissauer's tract, may also atrophy.

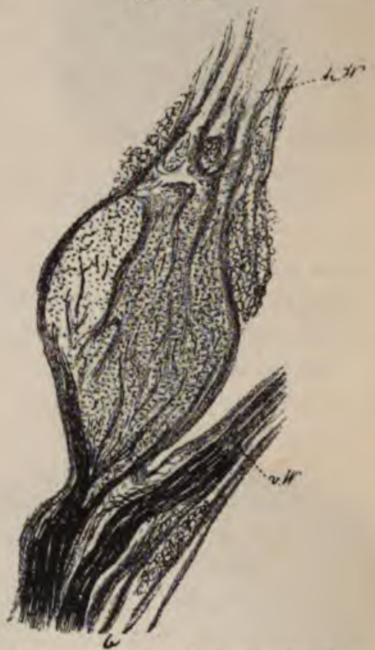
The posterior roots atrophy with great regularity. According to some (Leyden, Redlich, etc.), this atrophy is the primary lesion, and the spinal cord changes are secondary. It is, then, especially interesting to know that in some cases we were able to follow the disease of the posterior roots to the trophic centres, to the spinal ganglia, in which we observed an atrophy of the medullated fibres, as well as an atrophy of the ganglion cells. Sometimes the disease-process ascends to the medulla oblongata and produces atrophy of the *spinal trigeminal roots,* and often of the *solitary bundle* (Figs. 85*a* and 85*b*) and the posterior vagus nucleus; we never found the nucleus ambiguus diseased. Only rarely do other parts of the medulla oblongata, as the sensory trigeminal nucleus (Oppenheim), the motor (Raymond and Artaud), the nucleus of the auditory, and the hypoglossal nucleus (Raymond-Artaud, Koch-Marie) degenerate. In some cases the atrophy occurred in the nucleus of the nerves of the eye-muscles.

Fig. 84*a*.



Sagittal section through the spinal ganglion with the posterior and anterior roots and efferent nerves. (Weigert's stain.)

Fig. 84*b*.



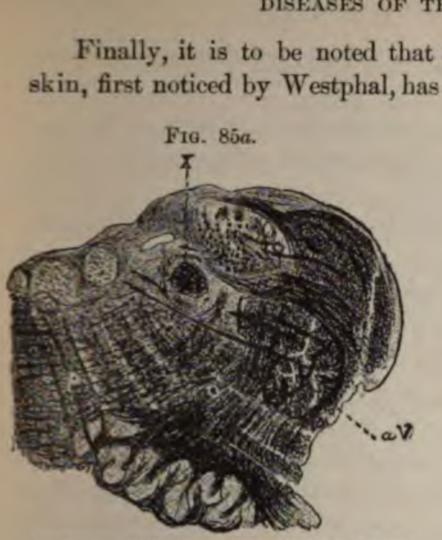
(Compare with Fig. 84*a*.) Atrophy of the posterior root and of the spinal ganglion in tabes dorsalis. *h. W,* posterior root. (Weigert's stain.)

The optic nerve becomes involved more frequently than the other cranial nerves. *Gray degeneration of the optic nerves* is often observed. The nerves of the ocular muscles may also become atrophied without their nuclei being affected. The same holds true for the vagus and recurrent laryngeal. A lingual hemiatrophy can also be of neuritic origin (Obersteiner, Cassirer, and Schiff).

Fig. 86*b* shows a degeneration of the Gasserian ganglion, the cells and fibres being involved. This discovery is important, because the Gasserian ganglion is the origin of the spinal trigeminal root.

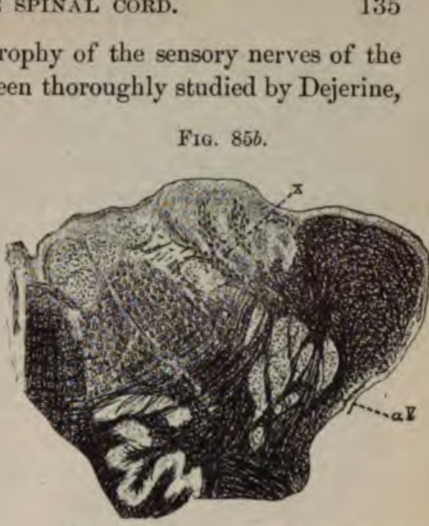
Finally, it is to be noted that atrophy of the sensory nerves of the skin, first noticed by Westphal, has been thoroughly studied by Dejerine, Siemerling, and myself, and lately by Gumpertz, from pieces of skin taken from living patients. The sensory nerves of the skin of the lower limbs are especially involved.

FIG. 85*a*.



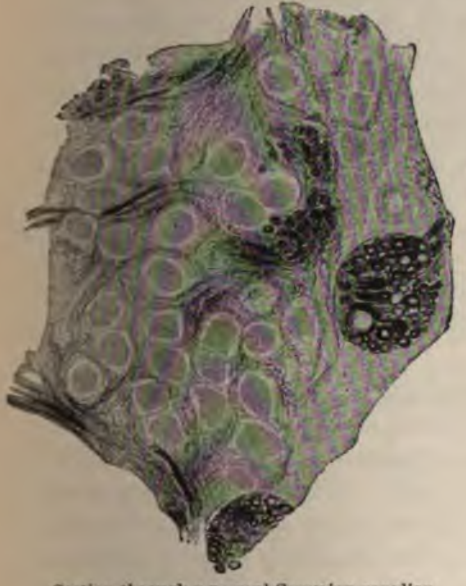
(Compare with Fig. 85*b*.) Part of a cross-section through the medulla oblongata at the height of the twelfth and tenth cranial nerves. *X*, solitary fasciculus; *a V*, spinal trigeminal root. Normal. (Weigert's stain.)

FIG. 85*b*.



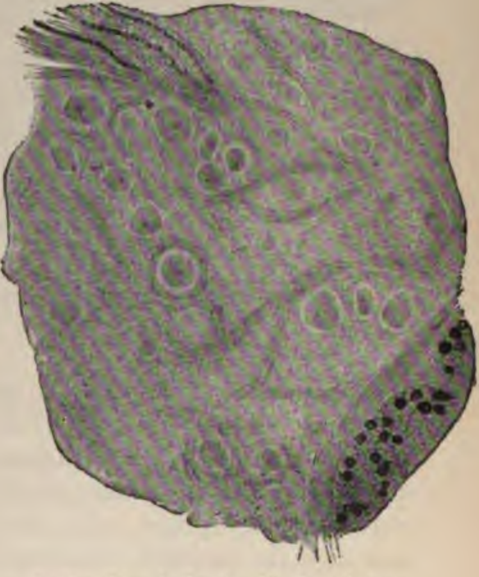
Part of a cross-section through the medulla oblongata at the height of the twelfth and tenth nerves. Atrophy of the solitary fasciculus *X* and of the spinal trigeminal root *a V*. Tabes dorsalis. (Weigert's stain.)

FIG. 86*a*.



Section through a normal Gasserian ganglion. Treated with osmic acid.

FIG. 86*b*.



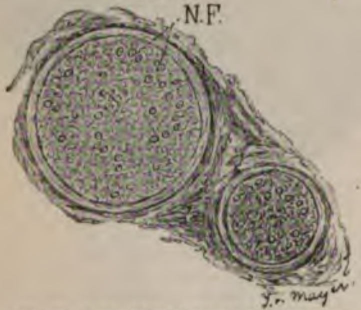
Section through the atrophic Gasserian ganglion in tabes dorsalis.

What part this plays in the symptomatology, whether it is primary or secondary, we do not know.

The observations of Dejerine have shown that a neuritis of the peripheral nerves may produce a symptom-complex very similar to that of tabes dorsalis (peripheral neuro-tabes).

The point of origin of tabes dorsalis has been sought for at different places. Two views held to-day are worthy of notice. The first was established by Marie, and modified by myself as a result of my observations, and may be formulated thus: The exciting cause of tabes acts upon the spinal ganglia and their homologues, and injures them without altering them structurally. This is sufficient to cause atrophy of the sensory fibres of the spinal cord, of the medulla, and of the periphery, which come from the ganglia. This atrophy gradually ascends—*i.e.*, towards the ganglion cells—until their fibres are also involved.

FIG. 87.



Atrophic peripheral nerve on cross-section. N.F., normal fibres.

The second hypothesis also accepts an exogenous origin for tabes, and supposes it to commence in the posterior roots. Obersteiner and Redlich have noticed that the posterior roots decrease in size as they pass through the pia mater, and conclude that every meningeal affection which produces thickening and shrivelling will injure the roots and cause them to atrophy. Many facts, however, contradict this view. Meningitis is not always found, it does not explain the involvement of the cranial nerves, etc. Recent discoveries, which show that diseases of the *peripheral* nerves may evoke diseases of the spinal ganglia, and even of the posterior roots and tracts, bring up anew the possibility of a peripheral origin for tabes.

Course and Prognosis.—A chronic disease. Its duration averages ten years; but it may last twenty to twenty-five years, and even longer. If the ataxia and bladder disturbance develop early and decrease quickly in intensity, death may ensue in a few years. When optic atrophy is an early symptom, we may expect the disease to last a long time. In the final stage the patient is bed-ridden. The emaciation becomes extreme. The movements of the legs, by reason of the general weakness, the long-continued inactivity, and the secondary alterations, are very limited. A real paraplegia, however, rarely occurs. The patient dies from marasmus, cystitis, and pyelonephritis, infection resulting from decubitus or from any intercurrent disease.

The prognosis is unfavorable. Recovery is rare. Oftentimes, however, the patient remains in an early—and bearable—stage of the disease.

The prognosis of individual cases may be influenced by the character of the symptoms. If the lightning pains are very severe, and if the attacks occur at very short intervals, the life of the patient is burdensome and his ability to work considerably lessened. The same is true of the gastric crises. In contrast to these are those patients whose symptoms are only of diagnostic importance and do not influence the patients' ability to work or to enjoy life. I treated a man who has had moderate lancinating pains for thirty years, yet who still goes daily to his business; another patient, whose lightning pains commenced in 1870, is still, notwithstanding his tabes, steady on his feet, and seemingly well and hearty. A third who had lues forty years ago, and in whom pupillary dilatation occurred twenty years ago, has to-day no symptoms outside of the Argyll-Robertson pupil, the Westphal's sign, and the bladder paresis.

Some symptoms gradually disappear, even though the disease progresses. I have seen a complete ophthalmoplegia which occurred at the beginning of an attack almost completely recede. The bladder disturbance is also often transient. The crises of vomiting—and, according to Charcot, the laryngeal crises—may disappear as the disease progresses.

Treatment.—Efforts to cure the patient or better his condition, or to still or quiet his subjective troubles, have led to the use of many methods of treatment. Occasionally it is possible to improve the patient's condition and often to reduce the pain and misery.

The following have all been used and recommended:

1. DRUGS.—Nitrate of silver in doses of one centigram ($\frac{3}{20}$ gr.), in pill form. This should be given for months, and after a short interval can again be given. Not more than ten grams (ʒijss) should, however, be allowed to be taken, on account of the danger of argyria. Also ergot (0.3, gr. ivss, as a dose), potassium iodide, gold and sodium chloride (0.003–0.02, as a dose, $\frac{9}{200}-\frac{3}{10}$ grain), and mercury, in the form of inunctions or subcutaneous injections, may all be tried.

To reduce the pain:

Sodium salicylate	15–45 gr.	(1.0–3.0) a dose	To be given only under direction of the physician.
Antifebrin	4–8 gr.	(0.25–0.5) a dose	
Antipyrin	8–15 gr.	(0.5–1.0) a dose	
Phenacetin	8–15 gr.	(0.5–1.0) a dose	
Methylene blue	$1\frac{1}{2}$ gr.	(0.1) a dose	
Analgen	15–30 gr.	(1.0–2.0) a dose	
Salipyrin Lactophenin	8–15 gr.	(0.5–1.0) a dose	
Pyramidon	3–$4\frac{1}{2}$ gr.	(0.2–0.3) a dose	
Morphine	$\frac{3}{20}$–$\frac{3}{10}$ gr.	(0.01–0.02) a dose	
Codeine	$\frac{3}{20}$–$\frac{3}{4}$ gr.	(0.01–0.05) a dose	

For the attacks of vomiting: Morphine and cerium oxalate.

2. ELECTRICITY.—Galvanic currents are used. The large electrode is placed on the nucha, the other in the lumbar region. The current strength should be of from five to eight milliamperes. The electrodes are kept at the same place, or the upper one may be moved down the spinal column. This treatment should be continued for months, daily or three times in the week, each sitting being of about five minutes. Stabile galvanic currents to the nerves of the legs are also recommended.

The Faradic Brush.—The large electrode is placed on the dorsal or cervical segment of the cord, while the other is placed over the anesthetic zones of the skin. Use a current strong enough to induce pain but not muscular contractions. Each sitting to last about ten minutes.

Attempts to influence other symptoms, as the gastric crises, by electricity have failed.

3. HYDROTHERAPY.—The mild carbonic-acid springs are the best for tabetics. Simple warm baths, as well as cold spongings, may also be tried.

4. MECHANICAL TREATMENT.—Operative stretching of a nerve is not used any longer. Suspension, massage, and gymnastic exercises, as well as bloodless nerve extension, are used.

Bloodless extension of the nerves is used, according to Bonuzzi and Benedict, in the following manner: The legs of the recumbent patient are tied together and drawn up over the head as far as they can be extended, so that by bending the body forward the knee is brought to the forehead or further. One must be careful, however, and remember the possibility of friable bones.

More mild is Blondel's method, who approaches the chin to the flexed knee and fastens it in this position for about five minutes by a band around the neck and knee-joint.

Another method of "stretching the cord through lengthening the vertebral column" is recommended by Gilles de la Tourette and Chipault. The legs of the patient, who is seated on a table, are held in an extended and adducted position, the upper part of the body being forcibly flexed forward. This should last from eight to twelve minutes.

None of these methods has produced so much comment and interest as the one lately recommended by Frenkel. This consists in methodical exercises in which co-ordinated movements are gradually retaught to the patient (pulling against the muscles,—Leyden's compensatory exercises). The patient is made to pass from simple to complicated movements,—not those which require much muscular effort, but merely co-ordination. He is made to relearn through the control exercised by his eyes and the rest of his sensory apparatus the proper motor impulses which induce co-ordinated movements.

There have been many apparatuses recommended by Frenkel and others which need not be discussed here. Special attention should be given to exercise in standing and walking. That it is of much value is without question; that it may in some cases, however, not produce the results expected of it is also true, and I regard it as unfortunate that the daily press (of Germany) has heralded it as the new method for the cure of tabes.

PLAN OF TREATMENT.—If the patient comes to us at an early stage of the disease, do not picture to him the gloomy side of tabes, but tell him that unless he lives well and rationally his nervous trouble may extend to the spinal cord. He should guard against *cold* and *excessive corporeal exercise*. I have met persons who in the first stage of tabes, when no ataxia existed, went upon a mountain tour, and returned with a completely developed ataxia. One-half to one hour walks in a flat country may be allowed; all climbing should be forbidden. Drenchings or wettings and the persistent use of the ice-bag should be avoided. Sexual excesses should also be guarded against. The patient should become accustomed to urinate regularly at least four times a day, even though he does not experience a desire for it. The food must be nourishing, and anemia and emaciation should be counteracted. I saw tabes progress very rapidly in two patients who were vegetarians. Smoking and drinking should not be carried to excess. The galvanic current used as previously described almost always gives good results. Faradic currents are not always well borne by a tabetic, and should not be continued for too long a time. *Internal faradization of the sphincter vesicæ* gives good results in incontinence of the urine. Stintzing advises the external application of electrodes over the symphysis and perineum with a galvanic current of from ten to twenty milliamperes, or galvanofaradization.

A stay at one of the numerous springs is of benefit. The use of hot[1] and cold water, steam, and sea-baths should be forbidden.

The suspension treatment (Motschutkowsky) is not intended for advanced cases, or for those complicated by arterio-sclerosis and by bulbar symptoms, or in threatening dementia paralytica. The ataxia, pain, and impotence may be relieved by this method, even the ocular symptoms. The application of a corset to support the spinal cord I have seen do good, especially in relieving the girdle pains.

The most difficult question to answer is, *Should antisyphilitic remedies be used?*

Personally I have never seen any good results from their use. Erb, among others, however, warmly recommends them, and claims excellent results. In those cases in which there is a positive history of syphilis, or in which the treatment was incomplete, or in which we find syphilitic symptoms, or in which the disease is atypical, so that the possibility of syphilis of the spinal cord cannot be excluded, by all means use mercury. Erb had one patient to whom he had given four thousand grams of unguentum hydrargyri without any bad results. He recommends repeated treatments, each lasting a short time, and in the intervals using electricity, baths, and tonics (especially strychnine). The lancinating pains are especially important to treat. Combat them with wet-packs, chloroform liniments, dry cups, the faradic brush, and the static breeze. Generally, however, these methods do not achieve any good results, and we find it necessary to use drugs: sodium salicylate, antipyrin, antifebrin, phenacetin, and analgen. Morphine should be the last resort.

---

[1] I have had very bad results from the use of hot water in tabes, notwithstanding the views of a well-known authority as to its efficacy.

The attacks of vomiting are, as a rule, not prevented by any drugs, though morphine and cerium oxalate may be tried, and will often do a great deal of good. The chief indication is to strengthen the patient between the attacks, so that he may be able to compensate for the loss of strength during them. Charcot advises the use of the Paquelin cautery. Applications of cocaine are to be used for the laryngeal crises. Frenkel's method is the best to counteract the ataxia. For the joint-diseases an orthopedo-mechanical treatment — rarely puncture or arthrectomy — is advisable. Bloody operations are abhorred by most surgeons in such cases As a *prophylactic* procedure the energetic treatment of the syphilis is the best one, though this is not always a certain method.

SPASTIC SPINAL PARALYSIS (LATERAL SCLEROSIS).

This is clinically a well-defined disease, but its pathological anatomy is not well understood. We owe our knowledge of it almost entirely to Erb and Charcot. This disease is observed in both children and adults. We will discuss first the form seen in the latter.

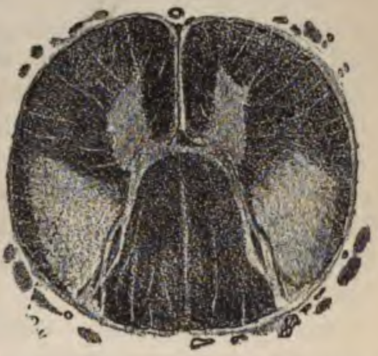
It is a chronic disease and attacks the lower limbs first. The symptoms at first are very slight. In protracted walking the patient tires soon and notices a feeling of *tension and stiffness* in the legs, "as if his tendons were too short." This is especially noticeable in complicated, difficult, and rapid movements of the legs, as in dancing, skating, hill-climbing, etc., and, as a rule, it attacks one leg more than the other. The weakness and stiffness increase slowly until observers also notice the motor disturbance. The first symptom found at this stage is the *increased knee reflexes*. At the same time, or soon thereafter, a *rigidity* is noticed with forced passive movements, as well as a motor weakness, which is very slight at first. The latter may, it is true, be absent a long time, the muscular rigidity alone preventing free active movements. Gradually these phenomena increase till a complete *spastic paresis* is present. The skin reflexes are also increased. The muscular tissues remain of normal volume and of normal electrical excitability. The gait becomes slower and slower, the toes are raised with more difficulty each week, until finally only very short steps can be taken. The rigidity then becomes so great that the examiner can hardly overcome it. Active movements are at times accompanied by a spastic tremor; it may be so great that the whole extremity remains in a condition of tonic spasm. There are no pains, or but slight ones, the *sensibility* is normal, and the *bladder* and *rectum* are not involved. *The spastic paralysis alone represents the clinical picture.*

The disease may spread to the arms, where the same symptoms would be found.

It may last a long time. Erb knows of cases which have persisted from ten to twenty years without any further symptoms. The disease may also come to a standstill.

In a majority of the cases which are thought to be spastic spinal paralysis, in the further course of the disease other symptoms arise from which we conclude that another disease of the nervous system (as multiple sclerosis, chronic myelitis, atrophic lateral sclerosis, hydrocephalus, etc.) is present. Always watch for errors; always be on the lookout *to unmask the spastic spinal paralysis;* every symptom which does not clearly belong to the spastic-paretic symptom-complex is an index to another disease. Multiple sclerosis, especially in the initial stage, which lasts for many years, is apt to clothe itself under the form of spastic spinal paralysis. There are, however, cases which must be classed as spastic spinal paralysis and nothing else, and it is advisable, therefore, to recognize it as a distinct entity.

FIG. 88.



Degeneration of the crossed pyramidal tracts. (Weigert's stain.)

Pathological Anatomy.—Before a case of this description would come to an autopsy we should expect to find a primary degeneration of the lateral tracts, especially the crossed pyramidal tracts. (Fig. 88.) In unmixed cases the patient remains alive a long time, and his symptoms are not severe enough to warrant a long treatment in a hospital. The cases which reach the post-mortem table are therefore clinically not pure, and so do not show simply a lateral sclerosis, but a complicated disease, in which, among other lesions, the crossed pyramidal tracts are found affected. But in some cases (those of Minkowski, Strümpell, and especially of Dejerine and Sottas) this was not the case; and these cases almost confirm in themselves the opinion that there is a *primary isolated lateral sclerosis which confines itself to the lateral tracts, especially the crossed pyramidal tracts, for a long time.*

Strümpell has made us conversant with an *hereditary familial type of spastic spinal paralysis.* It is found in the male members, is inherited, and commences between the twentieth and thirtieth years with a purely spastic disorder of movement of the legs, and results, as a rule, after many years in a real spastic paresis and paraplegia. The muscles of the arms, lips, and tongue are involved later and more rarely. It may last thirty to forty years, and in the last stage sensory disturbances, especially of the temperature sense, and slight weakness of the bladder are noticed. This makes it diverge from the symptom-complex of pure spastic spinal paralysis; and this difference showed itself anatomically in the one case examined, in that Gowers's tract and the direct cerebellar tract were degenerated in addition to the pyramidal tracts. This is, then, strictly speaking, probably a systemic disease, not a lateral sclerosis. Further, we must add that many of the hereditary and family spastic paralyses are accompanied by disease of the brain (see Little's Disease).

As already remarked, be guarded in diagnosing spastic paralysis. It may be the residuum of a myelitis or of a pressure myelitis. Compression of the spinal cord, as by tumors or meningeal or spinal-cord diseases, presents other symptoms,—deformity, hyperesthesia of the vertebral column, girdle-sensation, sensory disturbance or weakness of the bladder, etc., though the spastic paraparesis may be the first symptom of the compression. To distinguish it from multiple sclerosis, we must refer you to the discussion of that disease. Look for cerebral symptoms. If changes in the optic nerves are found ophthalmoscopically, all doubt is at rest. The detection of degenerative atrophy (first in the small muscles of the hand) guards against all confusion of amyotrophic with pure lateral sclerosis.

Hysteria may produce a spastic paralysis of the legs; but it is always acute and is a result of psychical excitement or an attack of spasm. The contracture possesses also all the characteristics of an hysterical one. In addition, other hysterical symptoms will be found. Cases of hysteroneurasthenia in which we find only weakness of the legs and increased knee reflexes are difficult to diagnose. False patellar and ankle clonus in such cases may also be found, often not distinguishable from the true. In these cases, however, the rigidity is absent; the paresis does not gradually progress, but suddenly disappears or is replaced by other symptoms.

Although weakness and increased tendon reflexes are often found in neurasthenia, still in such cases the rigidity and difficulty in passive movements are not present. It also more often occurs that a spastic spinal paralysis is mistakenly called an hysterical or neurasthenic condition than the contrary.

Age and Causation.—It commences generally between the twentieth and fortieth years. It has been traced to *syphilis*, several times to a *trauma*. It may develop in the *puerperium*, after *acute infectious fevers*, and as a result of lead intoxication. Poisoning with the different forms of lathyrus (especially sativus and cicera, from which a kind of bread is made in India and Algiers) may, according to the observations of Cantani, Bouchard, Proust, etc., produce a nervous disorder whose symptom-complex almost resembles that of spastic paraparesis.

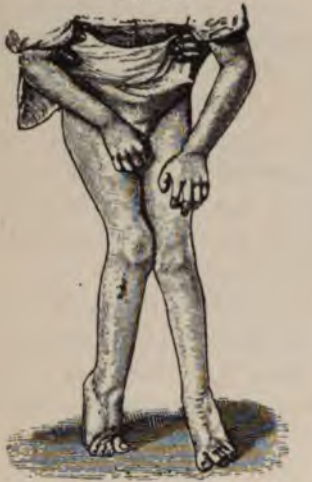
Muchin has observed toxic types, but nothing definite is known concerning them. We will discuss a syphilitic form in the chapter on spinal lues. The simple increase of the knee reflexes which is often found in arthritis is said to be liable to pass into spastic spinal paralysis.

CONGENITAL OR JUVENILE SPASTIC PARAPARESIS; CONGENITAL SPASTIC RIGIDITY OF THE LIMBS; LITTLE'S DISEASE.

This disease, although very similar to the paralyses of spinal origin, is in reality in most cases a cerebral disease in which the motor conducting paths of the spinal cord have become involved and therefore either become sclerosed or remain incompletely developed.

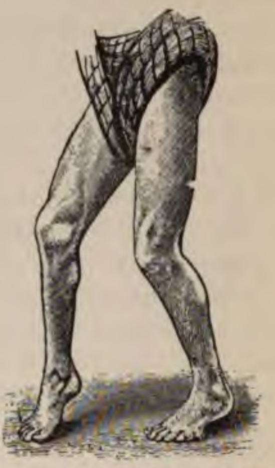
Though we regard it as a congenital malady, the muscular anomalies are not recognized immediately after birth. In a severe case, however, we do find soon afterwards that the thighs are pressed together, are hard to separate, and that after separation they return to their position of adduction. Generally, however, the condition of the child is only recognized upon attempts to walk, which he does later than usual. The gait is peculiar: the legs are held stiff, the thigh rotated inward and strongly adducted, so that the knees touch and rub each other in walking. This may be so strongly marked that the thighs cross each other in walking. The hip- and knee-joints are generally slightly flexed, the foot, however, being forcibly extended, so that the heel never reaches the ground, and the little one can merely drag himself along on the balls of his toes. (Figs. 89, 90.) The limb is moved as if it were one piece, and the pelvis alternately elevated and depressed. When the patient is reclining we can examine and note the rigidity and motor weakness which always occur, though not necessarily to the same degree, as one may be more marked than the other. The knee-jerk is always increased. In sitting the rigidity and the spasm of the extensors may be so great that the leg cannot be completely flexed, but sways in the air. Occasionally it may be impossible for the child to sit. The motor system is alone involved. The arms may or may not be affected. If they are, their position is peculiar. The upper arm is adducted, the lower arm flexed, the hand flexed or extended, at times pronated and the fingers flexed. It varies in different cases, but there is always something forced about it. Passive movements are difficult to execute. The tendon reflexes are increased. The movements are slow, awkward, and weakened. These pure types of spastic paresis of the lower or all four extremities correspond to spastic spinal paralysis and give a basis to the view that there exists an angenesis, a *developmental inhibition* of the *spinal pyramidal tracts*, or a degeneration of them. Often, however, we find other symptoms which make us think of a *cerebral origin* to the disease. (See chapter on Cerebral Palsies of Childhood.) These are,—

FIG. 89.



Station in simple spinal palsy of childhood. (After Seeligmüller.)

FIG. 90.



Gait in simple spinal palsy of childhood. (After Seeligmüller.)

1. *Strabismus.*—Concomitant "cross-eye" due to refractive anomalies or to a spastic condition of certain ocular muscles.

2. *Speech Disturbances.*—The muscular rigidity may attack the muscles of articulation and produce difficult and indistinct speech. It may, however, resemble that observed in bulbar lesions and be accompanied by difficult deglutition.

3. *Psychical Disorders.*—The loss of intelligence may amount to idiocy.

4. *Epilepsy* may occur soon after birth or in later life.

5. *Choreatic-Athetotic Movements.*—These may occur in all four extremities or in the face. This spastic-athetotic paraplegia must not be confused with chorea.

Etiology and Pathology.—Premature birth, difficult labor, use of forceps, or prolonged retention of the head high up before delivery may all be causes. Twins, inflammations of the motor zones during fetal life, and hereditary syphilis have all been considered as causative factors. Meningeal hemorrhage confined to the motor zone or to the leg-centre may be evoked by compression of the skull during birth. In those traumatic cases where no hemorrhage or material lesion occurs we assume that the pyramidal tract suffers most, as its fibres are still non-medullated. We express this by the words "*congenital spastic muscular rigidity.*" In other cases encephalitic processes, which produce atrophy, induration, or even *porencephaly,* are supposed to be the cause.

Inflammation and other diseases may, of course, attack the motor region of the cerebrum in childhood, but they are unilateral and produce a spastic infantile hemiplegia. They occasionally, however, attack both sides, and then give us a clinical picture very similar to that of Little's disease, but are not congenital.

Is there a congenital spastic paralysis of purely spinal origin? Although pathology does not give conclusive evidence of such a condition, van Gehuchten, Souques, Raymond, and Erb were induced to believe that such a condition existed; and Dejerine has lately been able in a typical case of this disease to show that it was the result of cervical degeneration, with secondary degeneration of the pyramidal tracts, which occurred in fetal life.

Prognosis.—The less severe the symptoms, the more it resembles spastic spinal paralysis, the better the prognosis. If spastic paresis of the legs exist without further symptoms, it may ameliorate in later life. The free use of the arms and a normal intelligence enable the patient to work in later life and reach old age. Some cases are so slight that the layman can not detect anything wrong. Involvement of the arms, combination with athetosis, psychic disorder, and epilepsy are all unfavorable signs.

Treatment.—Do not allow the child to attempt to walk at too early an age or to over-exert itself. Even in later years rest is imperatively demanded. Warm baths decrease the severity of the spasms, though the rigidity is lessened for only a short time. Massage, slight stroking and rubbing of the muscles, passive movements, gymnastics which act against the contractures, are all good procedures to be instituted. Tenotomy may first be necessary. Orthopedic treatment should follow the tenotomy, so as gradually to bring the extremity from its position of contracture. If the paralysis is marked, tenotomy will do no good.

Lorenz has lately recommended very strongly the surgical-orthopedic treatment of these conditions,—tenotomy, myotomy, myorrhexis, etc., forcible *rédressement*, even over-correction of the contracture. He has had some very good results, and in severe cases does not even stop at section of the obturator nerve. Drugs are not of much use, but the bromides, belladonna, hyoscine, potassium iodide, and solanin may be tried.

COMBINED DISEASE OF THE POSTERIOR AND LATERAL TRACTS OF THE SPINAL CORD.

Anatomy.—In typical cases of tabes the lesion of the white matter is confined to the posterior tracts. Rarely, as I found in one of my cases, are the direct cerebellar tracts attacked. In such cases we find not only that the fibres of Clarke's columns have disappeared, but also that the ganglion cells have degenerated.

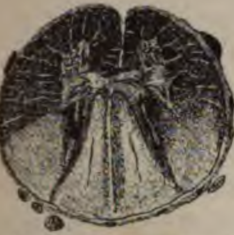
It, however, often occurs that different tracts of the cords, and especially the posterior and lateral tracts, are simultaneously attacked (Kahler and Pick, Westphal, Strümpell).

The disease is at times systematic,—*i.e.*, the different systems in the posterior and lateral tracts are affected, Goll's, Burdach's, the *direct cerebellar*, and the *crossed pyramidal*. Often, however, we do not find a systematic spreading of the process. (Figs. 91, 92, 93.)

Gowers's tract and the *anterior pyramidal tract* may also be attacked. If the disease does not confine itself distinctly to the different tracts, it may be confused with myelitis. From my own observations and those of others I have formed the opinion that combined systemic diseases may be confused with diffuse degenerative and inflammatory processes, but that such a condition undoubtedly exists.

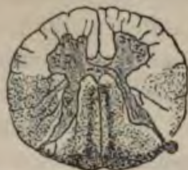
Symptomatology.—The symptoms are easy to describe when we consider the symptoms of isolated disease of the posterior tracts and isolated disease of the lateral tract, and then combine the two. But here we have a difficulty. Diseases of the posterior cord evoke atony of the muscles, Westphal's sign, and ataxia; and disease of the lateral tracts produces increase of the muscular tonus, increased tendon reflexes, and motor weakness. A combined disease of these tracts produces, therefore, symptoms which partly counteract each other. Westphal's observations have, however, disposed of the difficulty. He has shown that the symptoms found are dependent upon the intensity and diffuseness of the process in the two areas. If the direct pyramidal tracts are principally involved, and if the degeneration of the posterior tracts does not extend to the lumbar segment, the muscular tonus will be increased, and we shall find muscular rigidity, increased knee-jerks, and motor weakness; and the degeneration of the posterior tracts is shown by the presence of other tabetic symptoms, especially ataxia. A spastic-ataxic paraplegia or paraparesis which leads to a typical disorder of gait is the condition presented. Other tabetic symptoms may also occur,—bladder disturbance, lancinating pains, and disorders of sensation, which may, however, be wanting. Pupillary rigidity and optic atrophy are rarely observed.

FIG. 91.



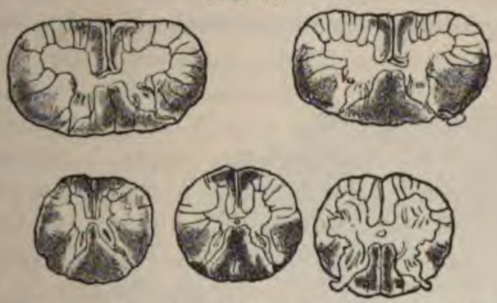
Combined disease of the posterior and lateral tracts. (Weigert's stain.)

FIG. 92.



Combined disease of the posterior and lateral tracts. Irrregular extension of the degeneration. The degenerated parts shaded.

FIG. 93.



Cross-section at different heights of a spinal cord with combined disease of the crossed pyramidal, direct cerebellar, Goll's, Burdach's, and the direct pyramidal tracts. The degenerated parts are shaded.

If the degeneration of the posterior tract is marked, and if it extends into the lumbar cord, we find all the symptoms of tabes (Westphal's sign also); and only a motor weakness, which may increase to paralysis, gives evidence of involvement of the motor conducting paths.

It often occurs that in the beginning the symptoms of involvement of the lateral cords are more pronounced, and only later—when the degeneration of the posterior cord descends to the lumbar region—does the knee reflex become annulled, which until then had been increased, and atony replace the spasm. There are therefore two symptom-groups:

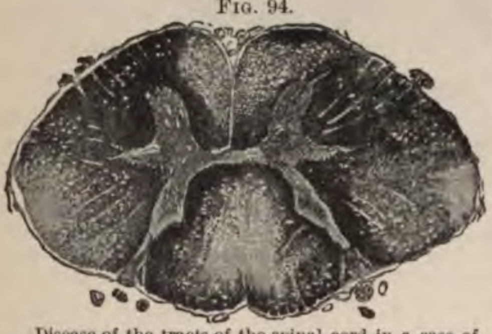
1. The symptom-complex of "spastic spinal paralysis" combined with ataxia, lancinating pains, bladder atony, pupillary rigidity, or other tabetic symptoms.

2. The symptom-complex of tabes, in which from the beginning motor weakness is combined with the ataxia.

It is particularly characteristic for the second condition to be developed upon the first. The disease is not always recognizable during the life of the patient. If the degeneration of the posterior cord is a slight one, it may simulate spastic spinal paralysis. More commonly it resembles myelitis. If we find lancinating pains and ataxia as prominent symptoms in a disease which otherwise resembles myelitis, we must immediately think of a combined sclerosis of the cord. If pupillary rigidity occurs in addition, regard the condition as certain (Oppenheim). Mayer has observed bulbar symptoms.

Multiple sclerosis and spinal or cerebrospinal lues must also be differentiated. The partial optic atrophy and the speech disturbance of multiple sclerosis are, however, quite different; but nystagmus and tremor similar to that of multiple sclerosis have been observed. See the chapter on spinal syphilis for the differential diagnosis between it and combined systemic disease.

FIG. 94.



Disease of the tracts of the spinal cord in a case of pernicious anemia. (After a Boedeker specimen in my collection.) (Pal-carmine stain.)

Concerning the etiology nothing certain is known. A *congenital* predisposition producing some motor weakness and slight resistance of certain tracts or neurons to injury is a factor in some cases. Syphilis plays a minor part. *Anemia* and spinal concussion are given as causes, as are also the cachexias. Lichtheim, Nonne, Minnich, v. Noorden, etc., have seen symptoms of tabes and of combined systemic disease appear in pernicious anemia, and have shown the presence of the corresponding spinal cord lesion, which they, however, do not regard as a systematic one. These authors believe it to be due to minute hemorrhages, *probably arising from the vessels*, which hemorrhages later unite, and thereby simulate a tract disease. It attacks the posterior cords, also the lateral tracts and the rest of the white matter. The gray matter is also involved at times. Whether it is a sequel of severe (lethal) anemia, or whether both disorders are caused by the action of some poison, is not known. Sometimes the anemia only develops in the course of the sickness (Bastianelli).

In these disorders we have generally paresthesia of all four extremities, pains, and objective disturbance of feeling. The general motor weakness and the paresis, with which the ataxia is often combined, are particularly prominent. Contrary to what is the case in tabes, the upper extremities are involved early, often at the same time as the lower. It is also more acute—rarely lasting several years—and undergoes marked remissions (Nonne, Bowman). The symptomatology is similar then to cases of combined degeneration of the posterior and lateral tracts, but pathologically it could with just as much propriety be classed with myelitis. Similar alterations occur in leukemia (Schultze, Nonne) and *carcinoma* (Oppenheim, Lubarsch).

In most of the cases of combined sclerosis which I have observed the anemia and the loss of strength were very marked. In one there was a tumor-cachexia, in another anemia from lactation.

In *pellagra* we may have symptoms of *ataxic paraplegia*, and degeneration of the posterior and lateral tracts (occasionally also the anterior horns) is found on post-mortèm examination. In *paralytic dementia* or *general paralysis of the insane*, we may also have the same spinal lesions; but death from the cerebral disorder generally occurs before they are fully developed. Marie attempts to assign the process in this disease to a primary degeneration of the gray matter (atrophy of the columnar cells), but this is doubtful.

Course and Prognosis.—It is a chronic or subacute disease, may run its course in a few months, and rarely lasts longer than one or two years (excluding Strümpell's form). Its prognosis is bad, but in occasional cases recovery may be possible.

Treatment.—The treatment is similar to that of tabes and spastic spinal paralysis; but special attention should be given to the anemia and cachexia which are generally present.

HEREDITARY ATAXIA (FRIEDREICH'S DISEASE)

is rare. It is a family disease, several members of a family usually being affected: it is but seldom that only one person is stricken. Direct heredity is rare, but epilepsy and cerebral diseases have been observed in blood relations. Consanguinity of the parents was observed once. It commences in childhood, in the seventh or eighth year, or during puberty, rarely later. Hirschl cites a case in which it followed an acute infectious disease, as do others.

The first symptom is an *ataxia* of the lower limbs. The patient walks with legs spread apart, with stamping,—but not so markedly as in tabes,—and the gait is unsteady and swaying. Charcot designated the gait as *démarche tabéto-cérébelleuse*, because the uncertainty has some similarity to that of cerebellar incoördination. In standing it is also very marked, not increasing, however, with closed eyes. The ataxia appears also in a reclining position, not only with movements, but also in attempts to keep a certain position, as in sitting, etc. (static ataxia). It progresses slowly, and gradually embraces the upper extremities, and occasionally the whole body. Active movements of the arms may at times excite ataxia and tremor simultaneously.

A slight muscular weakness may accompany the ataxia, but it is never considerable or general; only in the later stages does it reach a high degree, and may then be combined with contracture and atrophy. In most cases choreiform movements accompany the ataxia; at times also fibrillary twitchings in the different muscles, which are independent of the will. When the patient stands or walks we may observe a nodding and wabbling of the head. A distinction between the choreiform twitchings and the static ataxia is difficult to make.

Although the knee reflexes are annulled early, there is little or no sensory disturbance. Friedreich, however, showed that decreased sensibility may occur in the last stage. Crises and atony of the bladder never occur.

Nystagmus and *speech-disorders* are typical symptoms. The speech is slow, labored, indistinct, of irregular and mangled articulation; some words or syllables are lengthened, others spoken sharply. *Vertigo* occasionally occurs. Pupillary rigidity and paralysis of the ocular muscles do not belong to the symptom-complex of Friedreich's disease, although they may occur. There is no disturbance of the memory; the intelligence is generally normal. Scoliosis often occurs. A pes equino-varus with a marked hyperextension of the toes has been noticed in some cases (Fig. 95). One of my patients suffered with hypospadia and micrognathia. Other deformities may also occur.

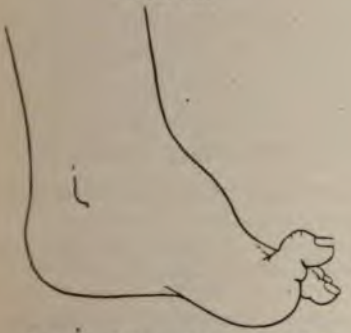
It is a very chronic disease and may last twenty to forty years, the locomotion being more and more affected until the patient is confined to his bed and death results from some intercurrent affection. The prognosis is bad. No case has ever been cured.

Pathology.—The spinal cord is throughout smaller than normal; we have also a *combined disease of the posterior and lateral tracts* (Schultze), a degeneration of Goll's tract *in toto*, of Burdach's almost entirely, and of the direct cerebellar, the crossed pyramidal (?), and of Clarke's columns, in which we find not only atrophy of fibres, but also a degeneration of the ganglion cells. Gowers's tract may likewise be involved.

Recent observations (Menzel, Nonne, Auscher) show that atrophy and developmental inhibition of the cerebellum may result in a symptom-complex which is closely allied to Friedreich's disease.

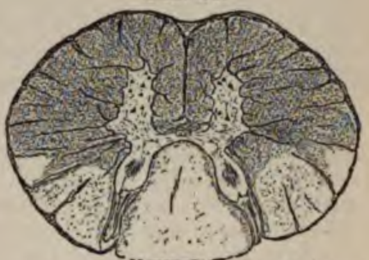
Senator believes that a congenital atrophy of the cerebellum is the basis of the disease. Marie has, however, established a symptom-complex distinct from Friedreich's disease which he calls *cerebellar hereditary ataxia*, and which he thinks results from developmental inhibition of the cerebellum. He includes therein the cases of Nonne, Menzel, Fraser, Klippel-Durante, etc., and adduces the following peculiarities: onset after the twentieth year, normal or increased tendon reflexes, paralysis of the ocular muscles, pupillary rigidity, disorders of sight and of the optic nerve, and occasionally disorders of deglutition. The scoliosis and other deformities are wanting. I doubt whether this differentiation can be completely made. Spiller has lately described a case examined anatomically.

FIG. 95.



Position of the great toe and condition of the foot in Friedreich's disease. (After Brissaud.)

FIG. 96.



Cross-section of the spinal cord in Friedreich's disease. Pal's stain. (After a Marinesco specimen in my collection.)

Multiple sclerosis, when occurring in children, offers the most difficulty for *differential diagnosis*. It does not, however, commence with ataxia, or, if it does start thus, spastic paresis, with increased deep reflexes, occurs at the same time. The optic atrophy of sclerosis is absent in hereditary ataxia, etc.

Hereditary syphilis may produce conditions hard to differentiate from Friedreich's disease. The acute onset, the remissions, the frequent optic and ocular paralyses, the spastic disorders, the apoplectic and epileptiform attacks, etc., offer, generally, a certain means for distinguishing cerebro-spinal lues from hereditary ataxia.

Treatment.—Careful nursing and avoidance of all injurious things. For treatment of the ataxia, see page 138.

POLIOMYELITIS ANTERIOR ACUTA; ACUTE ATROPHIC SPINAL PARALYSIS; SPINAL PALSY OF CHILDREN.

This disease, first described by Heine in 1840, is rare before the fifth month, and less so between the sixth and eighth; but most of the cases occur between the second and third years of life. After the fourth year, again, it occurs very rarely, although even adults are not exempt.

Cold and trauma have been given as *causes*, but more and more are we veering to the view that it is an infectious disorder. Its development and course remind one of an acute infectious disease; it occurs at definite times and in *epidemics*. A Stockholm physician, Medin, has reported an epidemic in which forty-four cases of this disease occurred in one month in the same town. It has seemed to me that most of my cases came from Weissensee, a suburb of Berlin. It often develops after other infectious diseases, especially measles, scarlet fever, and whooping-cough.

By the introduction of micro-organisms into the bodies of animals, Vincent and Bianchi have produced alterations in the cord which in localization and character resemble those of acute poliomyelitis. Crocq, Henriquez, and Hallion have done the same. A similar change has been observed in chronic sulfonal poisoning (Helwig).

Symptomatology.—The disease is ushered in abruptly with a high fever (102° to 104° F.) and with vomiting, anorexia, stupor, even coma and delirium, and occasionally convulsions. This stage of fever lasts from a few hours to some days, during which nothing shows the true character of the disease. Near or at its close the *paralysis* is noticed,—a paralysis *which is completely developed from the beginning*. Sometimes the initial stage is so short that it is overlooked, or it may be altogether absent; the child that lay down well awakes in the morning with a paralysis. One leg, more rarely an arm, or both legs are generally involved. Only in a few cases do we find a paralysis of both legs and one arm, or of all four limbs, or of an arm and a leg of one side.

Duchenne and Seeligmüller have furnished the following tables:

	Duchenne. Cases.	Seeligmüller. Cases.
Paralysis of one leg	32	42
Paralysis of both legs	9	14
Paralysis of one arm	10	13
Paralysis of the four limbs	5	2
Paralysis of both arms	2	1
Crossed paralysis	3	2
Hemiplegic paralysis	0	1

The paralysis is always *flaccid*, always a DEGENERATIVE one; is combined with *atrophy*, which is always shown by the *reaction of degeneration*.

The picture soon changes. Little by little some of the paralyzed muscles obtain a slight degree of mobility; the whole extremity is not removed from the influence of the will, but only certain muscular groups remain continually paralyzed. Take, for example, a common case, in which only one leg is attacked. Movement may return to all muscular groups except the extensors of the foot and toes (of these the tibialis anticus may be spared). The extensors of the leg may, again, be the only muscles involved. The sartorius, however, is generally intact. In the arms it is especially the deltoid, alone or in combination with the biceps, internal brachial, and supinator longus, etc., which is the seat of the chronic paralysis. In fact, all different combinations of muscles and muscular groups may remain paralyzed or recover. The cranial nerves, and even the nuclei of the motor cranial nerves, have in some cases been involved. The *concentration of the paralysis* lasts only a few weeks. Those muscles in which the faradic excitability is not lost after one week or in which it is soon afterwards present will recover, although the recovery may be very slow. The flaccid-degenerative paralysis is the feature of this disease; all else is subordinate to it. The knee reflexes are lost in the affected members, provided the quadriceps is persistently paralyzed. The same is true of the other reflexes. There is no pain, generally; rarely, it may be so acute as to simulate acute rheumatism. The muscles may be sensitive to pressure. If the pain of the muscles and nerves is considerable, some neuritis is probably present. The *sensibility is not decreased* except sometimes in the later stages, and is then probably a result of the decreased temperature of the skin. As a rule, the sensibility remains normal. The same is true of the *sphincters*. There is no psychical alteration. *Disorders of circulation, stunting of the growth, deformity*, and *stiffness of the joints* are secondary symptoms which may occur. The skin of the paralyzed member is cool and reddish-blue in color, and may differ 10° to 12° C. in temperature from that of the sound side. This is probably the result of vasomotor influence.

The paralyzed members do not grow normally; they become stunted, and in time show a marked shortening, from which results considerable difficulty in locomotion (Fig. 97).

Atrophy may also result (Fig. 98*a* and Fig. 98*b*). Paralytic contractures resulting from the influence of the antagonists occur often and are important. We find them most frequently in the foot. The most common form is pes equino-varus, which results from paralysis of the extensors of the foot and toes, especially when the tibialis anticus is not involved. If this muscle alone is atrophied, valgus ensues. If the muscles of the calf are paralyzed, pes calcaneus is produced, etc.

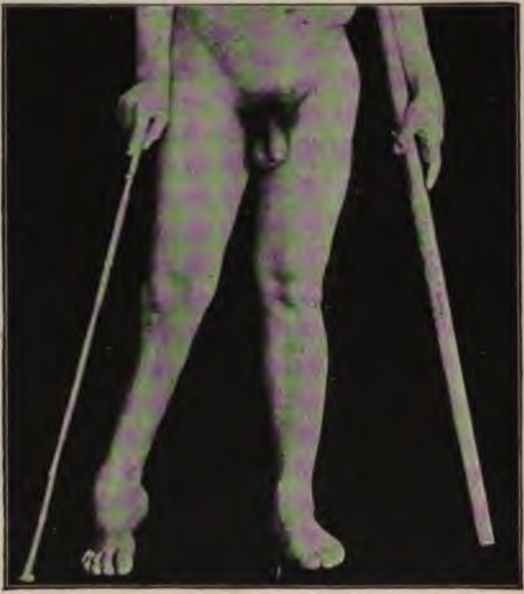
More rarely we find contractures in the upper limbs, the claw-hand being the most common.

Scoliosis and lordosis may also result from the atrophy produced by the muscular paralysis.

Looseness of the joints results when the muscles which support the joint become paralyzed.

Pathology.—The original lesion, first studied by Cornil, Prévost and Vulpian, Charcot and Joffroy, is an *acute inflammation* of the gray matter of the anterior horns. We find in a fresh case hyperemia of the anterior horns, dilatation of the arteries with hypertrophy, perhaps thrombosis, hemorrhage, and even leucocytosis. The round cells are especially heaped up around the vessels. (Figs. 99 and 100.) The nerve-fibres are swollen, the ganglion cells clouded, the nuclei indistinct, and the prolongations atrophied. This stage is succeeded by atrophy of the cells and fibres in parts of the gray matter. It may in the early stage extend over the neighboring parts of the white substance, and decreases as the disease continues. Analogous alterations have been observed in the nuclei of the medulla oblongata (Medin, Redlich). Most of the observations are from later stages, since death rarely results early. Circumscribed atrophy of the anterior horns with decrease in size of the same is generally found. (Fig. 101.)

FIG. 97.



Shortening and atrophy of the right leg. Pes equino-varus resulting from an acute anterior poliomyelitis in childhood.

FIG. 98*a*.

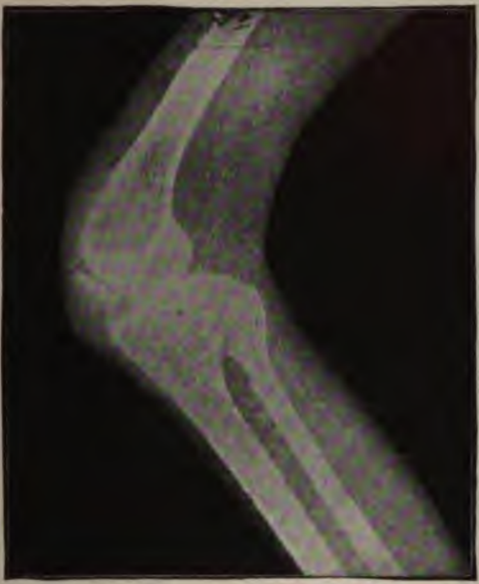
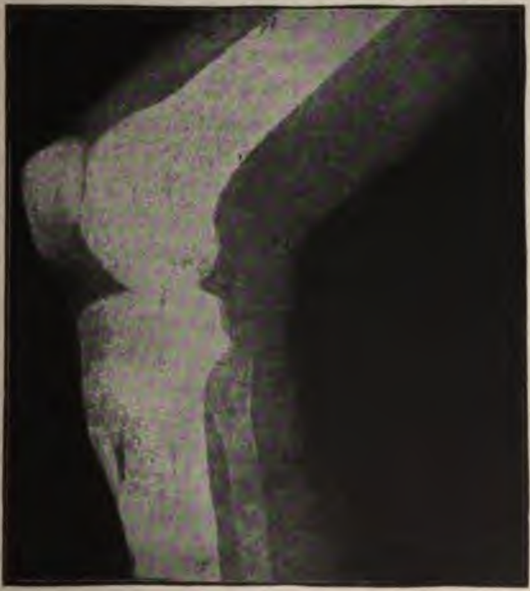


FIG. 98*b*.

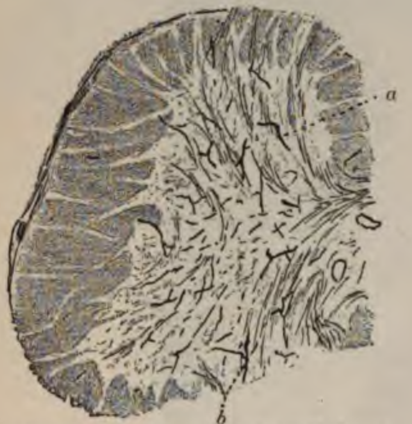


Radiographs of the lower extremities. Fig. 98*a*, in a case of spinal palsy of childhood; Fig. 98*b*, of a normal individual with limb in same position. (After Achard-Lévi.)

Marie sets up the hypothesis that infectious material (an infectious embolus) reaches the anterior spinal artery and the commissural branch thereof which passes to the anterior horn.

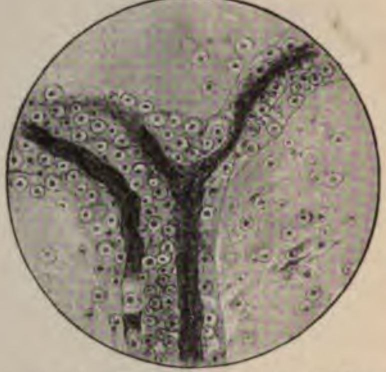
Differential Diagnosis.—Errors are especially liable to be made in the first stage. The fever, with pain (causing impairment of motility), may make one think of acute rachitis, coxitis, osteomyelitis, syphilitic pseudoparalysis, etc.

FIG. 99.



Acute anterior poliomyelitis (magnified very much). *a*, blood-vessel; *b*, blood-vessel.

FIG. 100.



Round-cell proliferation in the neighborhood of the blood-vessels in acute anterior poliomyelitis.

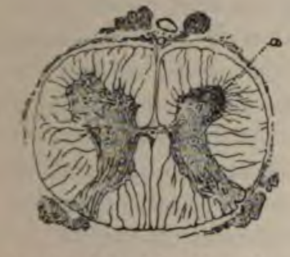
We can, however, always detect a muscular contraction when we try to produce passive movements, the little one endeavoring to hold his limb in the position in which least pain is experienced. The pain on pressure and in passive movements also indicates this disease. The electrical examination and the reflexes also help in the diagnosis.

It is more difficult to distinguish acute anterior poliomyelitis from *multiple neuritis*, as the latter does occur in children, though rarely. (1) In multiple neuritis the disease progresses more slowly, taking days or weeks to reach its acme, while in acute anterior poliomyelitis it has reached its height from the beginning. (2) The fever lasts longer in multiple neuritis. (3) The pain on movement and other pains are more severe and of longer duration, and the nerves are swollen and more sensitive to touch. (4) Disorders of sensation are almost always found with neuritis. (5) Edema is more often found with neuritis. (6) Involvement of the cranial nerves in doubtful cases speaks more for multiple neuritis. In some cases of poliomyelitis the peripheral nerves may become affected, and spinal changes may also occur in polyneuritis, thus making diagnosis difficult.

Cerebral and *spinal* palsy of childhood are easily distinguished from each other. I once diagnosed an encephalitis pontis in which further observation showed that it was a combination of an anterior cervical and lumbar poliomyelitis of the left side with polioencephalitis of the left nucleus of the facial nerve. This uncommon localization produced a picture of alternating hemiplegia; but the paralysis of the legs soon disappeared, while that of the arms was a flaccid degenerative one and concentrated itself upon Erb's muscles.

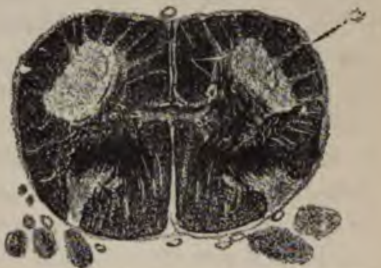
I also observed a case of acute disseminated myelo-encephalitis in childhood, in which some of the symptoms resembled a cervical poliomyelitis. The symptoms of acute atrophic spinal paralysis often follow so closely after a trauma that the possibility of a hemorrhage of the cord cannot be disposed of. *Hematomyelia* may indeed resemble in every particular an acute anterior poliomyelitis. Paralysis of the newborn is also a degenerative, flaccid paralysis, and involves in most cases only the arm, and here especially the region of the fifth and sixth cervical nerves.

Fig. 101.



Atrophy of the right anterior horn after acute lumbar anterior poliomyelitis. (Carmine stain.)

Fig. 102.



Bilateral acute lumbar anterior poliomyelitis. Terminal stage. (Weigert's stain.)

Acute myelitis is rare in early childhood. If bladder weakness, sensory disturbances, spasms, and ataxia occur, no matter if the atrophic paralysis is the chief symptom, it is a myelitis.

The slow development and combination of atrophy with sensory disturbance will differentiate a *lumbar spinal gliosis* from poliomyelitis.

Congenital muscular defects can easily be recognized.

Prognosis.—Good *quoad vitam.* Only in the initial stage has death been observed. Complete recovery can occur in only a few cases, but improvement is always possible. Those muscles in which after two or three weeks the faradic irritability is not lost will recover. The muscular groups in which after the first week we find a reaction of degeneration will remain paralyzed, though some improvement is possible in them. The secondary alterations help to produce loss of function.

Children who have had spinal palsy are predisposed to atrophic paralyses in later life.

Treatment.—Seek to prevent the progress of the process. *Absolute rest* is imperatively demanded; keep the bowels open, slightly bleed the child (leeches), and induce free perspiration, not with baths, but by the use of hot drinks and warm, heavy blankets. Do not use an ice-bag. Salicylates may be given, also belladonna, in the initial stage.

As a rule, however, the children only come under our treatment after the paralysis is complete and well defined. Then *electrical* and *mechanical* treatment is the right one. The children should, however, be kept in bed, and only be allowed to get up when movement does not increase the deformity. As soon as active movement has been reinstituted in a part of the affected muscles, strengthen them with electricity, massage, and gymnastics. One can commence after the second or third week. Use a galvanic current, the cathode upon an indifferent place or upon the diseased part of the cord, and with the positive pole stimulate the diseased muscles. Commence with a weak current, so as not to alarm the child, and increase until muscular contractions ensue. The muscles which react directly or indirectly to the faradic current may be stimulated with it.

Gymnastic exercises should include the muscles which have again recovered their function. A slight resistance to the movements may be made. Massage promotes the circulation and the nutrition of the muscles. At first only rub or stroke the muscles, afterwards they may be kneaded. Passive movement must be early instituted, to guard against contractures setting in. If necessary use bandages and specially-devised apparatus for this purpose; diachylon plaster applied in strips may be all that is necessary.

Warm baths of 90° to 95° F., with the addition of salt or other medicinal substances, are of benefit. Subcutaneous injections of strychnine have been recommended to counteract the paralysis.

For the deformity, tenotomy of the shortened tendons (or section of the fascia) is necessary when extension apparatuses fail. All apparatus must fit well, must not restrict the circulation or cause pressure, and must be modified as the limb grows. Arthrodesis of the ankle-joint to an extended position is recommended by Karewski when the leg is greatly shortened.

For methods of surgical orthopedic treatment, see works on surgery and orthopedics.

Drobnik has proposed a noteworthy method of transplantation or connection of the muscles with intact functions with the tendons of the paralyzed ones. For example, the tendon of the normal sartorius is to be divided and sewed to the paralyzed quadriceps. A healthy tendon may also be split into two, and one part joined to the tendon of the paralyzed muscle.

ACUTE ANTERIOR POLIOMYELITIS OF THE ADULT; ACUTE ATROPHIC SPINAL PARALYSIS OF ADULTS.

This is a rare disease, yet has been observed clinically in a number of cases and also in a few autopsies. Generally those attacked are between twenty-five and thirty years old. It may follow the acute infectious diseases, especially measles, also the puerperium or occur in connection with it. In one of my cases a severe gonorrhea preceded the attack. It appears that a severe cold has the power to produce this disease. According to Rank's statistics, this was the case in twenty-five out of thirty-six cases. Over-exertion and trauma have also been held responsible. Once I observed an atrophic paralysis, probably spinal, result from prolonged chloroform narcosis.

As in the infantile form, it commences with elevation of temperature, the febrile stage lasting, however, from one to two weeks. Pain may be present, especially in the back. If severe pain occurs in the extremities, the peripheral nerves are involved, and in obscure cases multiple neuritis may be diagnosed.

After the fever has almost disappeared, paralysis ensues, which embraces a larger part of the body than in the infantile form. Both legs or arms, or all four extremities, may be involved. From four to six days after the onset, alterations in electrical irritability, the reaction of degeneration, will be found. Increased galvanic excitability lasts only a short time. In from a week to a month the paralysis decreases in extent, and many muscles or groups of muscles which had been completely paralyzed gradually regain their functions, though others, and often a whole limb, may remain chronically paralyzed. Complete recovery rarely occurs. In case it does occur, the probability is that there had been a mistake in diagnosis and that it was rather a case of multiple neuritis. Partial recovery may take place after a month, or it may be three months before the first sign of movement has commenced, and may continue a year or longer, till that degree of recovery is reached at which further development of the process ceases. Later the electrical excitability returns to normal.

Occasionally fatty deposits in the atrophied muscles, at least in a part of them, occur, so that a certain similarity to the pseudohypertrophic forms of progressive muscular atrophy results.

Diagnosis.—As before stated, guard against confusing it with multiple neuritis. Hematomyelia develops more rapidly and has no preliminary febrile stage. The sphincters are also involved in this affection. Compare also the chapter on Landry's Paralysis.

Prognosis as to life is good. Only when the respiratory muscles are involved does death result. Complete recovery need not be expected.

On account of its greater involvement, it has a more unfavorable prognosis than the infantile form ; though as the bones have reached their full development, and as deformities rarely occur, the chances for recovery of the functions of a paralyzed member are better. Even when in bed their lower limbs are almost immovable ; they may be able to walk with the aid of crutches together with the compensatory use of their pelvic muscles.

Treatment.—In regard to treatment, that which was said in the article on the infantile form is true here. In addition, ergotin can be used in combination with atropin.

℞ Ergotin, 10.0 (ʒ ijss) ;
Atropin, 0.02 (gr. $\frac{3}{10}$).
Sig.—Twice daily, one-third to one-half a syringeful subcutaneously.

SUBACUTE AND CHRONIC ANTERIOR POLIOMYELITIS ; SUBACUTE AND CHRONIC ATROPHIC SPINAL PARALYSIS.

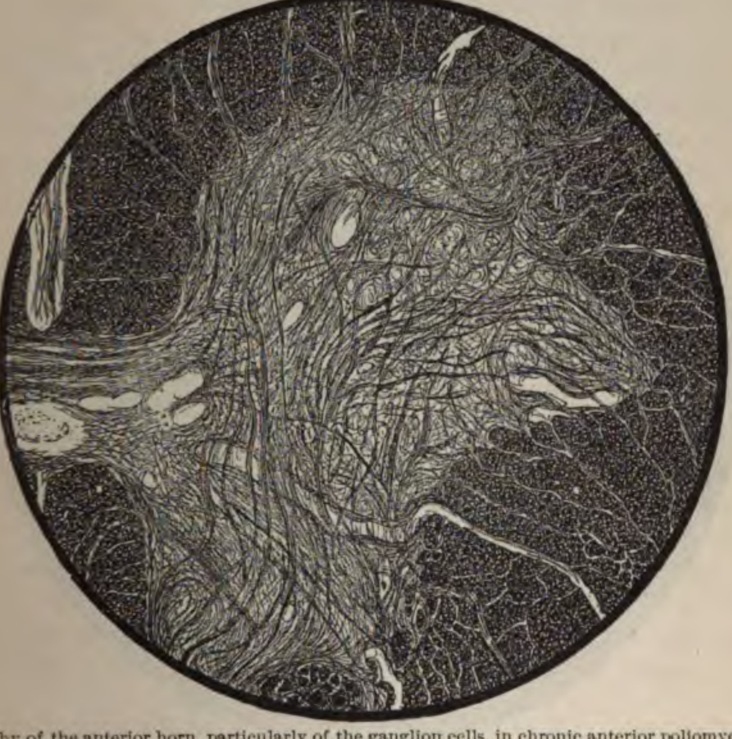
These rare forms of atrophic spinal paralysis occur late in life. The cause is unknown, though from their great similarity to the different types of lead-paralysis we surmise that some toxic substance is at the basis of the trouble.

Observations of chronic anterior poliomyelitis in the course of diabetes (Nonne) seemingly confirm this. On the other hand, Erb has lately laid stress upon traumata as a causative factor. Cases developing subacutely rarely occur ; its frequency was over-valued at the time when multiple neuritis had not been well studied, most of the cases observed falling in that category.

Cases of subacute and chronic poliomyelitis, however, occur, as post-mortem evidence has shown, and they can be clinically differentiated from multiple neuritis.

The patient feels a weakness in one leg, which increases and progresses day by day, the arms being attacked as often as the legs, until, after some months, both legs, arms, or all four limbs are to some extent paralyzed. If we were to examine the patient at this stage, we should find a flaccid paralysis, which has an *elective* character, as certain muscles or muscular groups are always unimpaired. In paralysis of the upper arm the triceps may remain free from it ; in paralysis of the whole arm the flexors of the fingers, the abductor pollicis, and other muscles of the fingers may remain intact; in the legs the peroneal region may be completely paralyzed, or with the exception of the tibialis anticus or peroneus longus, while the remaining muscles are not involved to any great extent. The paralysis is absolutely flaccid; the tendon reflexes in the region of the affected muscles are lost; and, furthermore, it is a *degenerative* paralysis. Atrophy follows the paralysis, and the reaction of degeneration is found present in a complete or incomplete form. A complete parallelism, however, between the paralysis and the degenerative reaction does not occur.

FIG. 103.



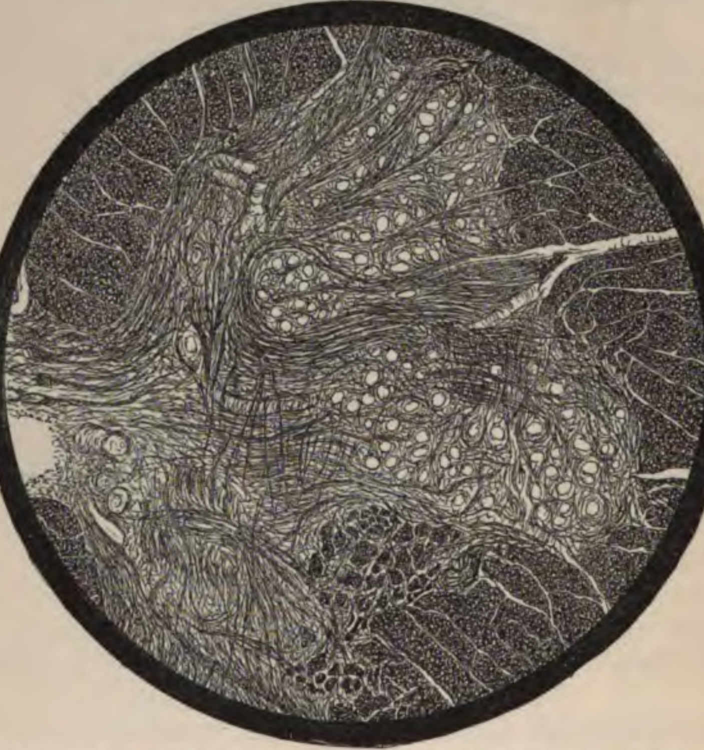
Atrophy of the anterior horn, particularly of the ganglion cells, in chronic anterior poliomyelitis.

Fibrillary twitching is almost always present. Sensibility in pure cases is intact. Slight rheumatic pains may be present in the beginning and during the course of the disease. Weakness of the bladder muscles does not occur, nor is there any sexual weakness. *Muscular paralysis* and *muscular degeneration* are the only symptoms. Different types are found. Some cases remain stationary, the paralysis neither progressing nor retrogressing after a certain point is reached. In other cases, after six to eight months the process decreases and partial recovery ensues. Again, some cases gradually increase in intensity until death results.

One muscular group after another becomes involved until all the four extremities and some of the muscles of the neck and trunk become affected. The patient lies motionless, without any pain and with no decubitus. The respiratory muscles become affected, and the patient dies from asphyxia, aspiration pneumonia, or some intercurrent disease.

Pathology.—Only a few cases have been accurately examined after death (by myself, Nonné, Charcot, and Dejerine). They reveal the presence of a primary chronic inflammation of the anterior horns, with atrophy and complete degeneration of the nervous elements—cells and fibres (Fig. 103; compare with Fig. 104). The white substance is intact, or perhaps a few fibres may have been involved in the neighborhood of the gray matter. It appears to be as much a disease of the anterior horns starting from the blood-vessels as it is a primary atrophy of the ganglion cells.

FIG. 104.



Normal anterior horn of the lumbar prominence. (Weigert's stain.) (Compare with Fig. 103.)

Differential Diagnosis.—The subacute form may be confused with multiple neuritis. The absence of ataxia, of sensory disturbance and of paralysis, of sensitiveness to pressure and swelling of the nerve-trunks, makes a diagnosis certain.

In chronic progressive cases amyotrophic lateral sclerosis, progressive muscular atrophy, and gliosis must be excluded. Absence of any spastic condition and loss of the deep reflexes speak for chronic poliomyelitis. Increased reflexes of the lower limbs, with a flaccid condition of the muscles of the rest of the body, as well as paralysis and atrophy of the small muscles of the hand, early weakness of the legs developing without any degenerative symptoms, marked bulbar symptoms,—all these are indicative of amyotrophic lateral sclerosis. Progressive muscular atrophy of spinal origin may be differentiated from chronic poliomyelitis *sensu strictiori* by the fact that the atrophy is the primary symptom and the paralysis only a result. The development also is slow and the atrophy attacks single muscles at a time, never a group of muscles. In most cases the small muscles of the hand are attacked first, while the poliomyelitis commences in the legs or the shoulder-muscles. The differentiation of these two affections, however, is an artificial one.

Gliosis is accompanied by sensory disturbances, and often with trophic alterations of the skin and muscles, and the atrophy develops slowly.

Prognosis.—The prognosis is always doubtful in reference to complete restitution. The quicker the paralysis limits itself, the more incomplete it is, the more favorable is the prognosis. The purer the case, the more positively neuritis can be excluded, the worse is the outlook for a complete recovery. The same considerations that were mentioned in regard to the acute form apply here.

Treatment.—In the first stage a diaphoretic treatment is indicated. Over-exertion is to be avoided. Electrical treatment should be instituted after the plan outlined for the acute form. Strychnine is of very little value. For further treatment, see chapter on Acute Poliomyelitis.

AMYOTROPHIC LATERAL SCLEROSIS.

Amyotrophic lateral sclerosis (Charcot) is a disease of middle life, having been observed in only a few cases in children. The cause is unknown,—cold, trauma, over-exertion, fright, have all been given as predisposing causes. In one of the cases observed by me the symptoms resulted after a severe fright, in which the patient was forced to row with all his strength for his life.

Symptoms.—In typical cases we find a combination of three symptom-complexes: those of chronic anterior poliomyelitis, of spastic spinal paralysis, and of bulbar paralysis. It is a chronic disease; commences with *weakness* and *atrophy* in the upper, or *weakness* and *stiffness* in the lower extremities. *Fibrillary twitchings* may precede the muscular atrophy and form the first symptom. The paralysis and atrophy spread and increase on the one hand, the muscular rigidity on the other hand, while other symptoms, especially pain, may be absent or but slight. In from six to eight months the arms and the legs may have become completely paretic.

Objective examination shows us at this stage about the following:

The upper arms are adducted, the lower arm flexed, the hands pronated, often in extreme flexion, the fingers becoming more or less fixed in the claw position. This results from muscular tension, active *contraction*, and the atrophic paralysis.

The *deep reflexes are increased*. A slight tap upon the tendons of the supinator longus or triceps, on the bony part of the lower arm or hand, produces marked muscular twitching; at times, tremor of the hand may be produced.

Symptoms which immediately catch the eye are *fibrillary tremor* and *muscular atrophy*. The latter commences more commonly in the small muscles of the hand; at the same time, or later, the shoulder or radial regions become involved. We also find complete or partial reaction of degeneration or simple quantitative decrease of the excitability.

Motor weakness is combined with the atrophy and contractions, a weakness which is not merely a result of the latter, but is an independent symptom.

The gait is normal for a long time, but at length becomes typically altered. The patient walks slowly, with stiff legs and by small strides, and afterwards can only push himself feebly forward on his toes. After one or two years he becomes bed-ridden, atrophy of the legs coming on, but not of so severe a form as in the upper extremities.

Pain is absent. A slight *paresthesia,* however, is often present. There are no other symptoms observed. In short, *atrophy, rigidity,* and *paresis* are the *only symptoms.*

Later, symptoms of *bulbar paralysis* occur; though they may occasionally come on in the first stage. The patient speaks indistinctly, with a nasal tone, which increases to *dysarthria*, and at last anarthria. Difficulty in deglutition occurs at the same time or later, also other symptoms indicative of paralysis of the muscles of the lips, tongue, palate, or cheek. A *masseter clonus* may be the first symptom of bulbar trouble noticed, being caused by the rigidity and increased tonus which may precede the paralysis. The atrophy develops later; the lips become thin; the tongue lies sluggishly on the floor of the mouth, is shrunken, trembles, and feels sponge-like. Partial reaction of degeneration may be found at this time.

In the last stage the lower half of the face becomes rigid, the mouth open, saliva oozes from between the lips, the angles of the mouth are depressed, the lips cannot be approached for whistling, a light cannot be blown out, the lower jaw is sunken, the tongue cannot be protruded, speech is blurred, and of a " lalling" character, swallowing is impossible, and *aphonia* is present. The uvula cannot be elevated. A laryngoscopical examination shows paresis of the adductors, etc. The patient laughs or cries easily; the paralysis of the extremities progresses more and more, and the rigidity decreases as the atrophy spreads.

Even in this stage all sensory functions are unimpaired. Asphyxia, inanition, or aspiration pneumonia hastens the end.

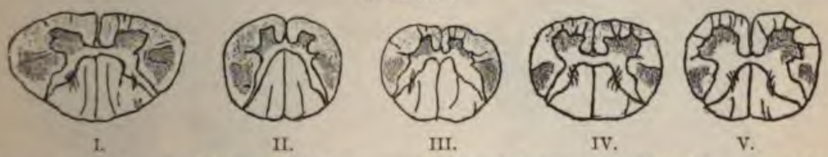
The disease lasts from two to four years, as a rule.

MODIFICATIONS.—In some cases the spastic conditions may be very slight. If they disappear entirely, the diagnosis of amyotrophic lateral sclerosis, in a clinical sense of the word, cannot be made. We speak then of a chronic atrophic spinal paralysis with bulbar paralysis, although the anatomical examination may correspond to an amyotrophic lateral sclerosis. It is probable that in such cases the process in the gray matter precedes that in the white substance a long time.

A rare modification is that in which there is little atrophy and only a spastic condition is in evidence. I have observed an acute development in one case.

Pathology.—The motor conducting paths and the trophic centres of the muscles are degenerated,—*i.e.*, we find an *atrophy of the pyramidal tracts* and of the *anterior horns.* The crossed pyramidal tract is especially involved, also generally the anterior pyramidal tract; and we often find a slight diffuse degeneration of the rest of the anterior lateral tracts. (Fig. 105.) The sensory paths are not affected. The anterior horns are the only part of the gray matter that is affected. Both cells and fibres are degenerated, only some of the reflex collaterals are intact. (Fig. 106.) The *anterior roots* are also atrophied.

FIG. 105.

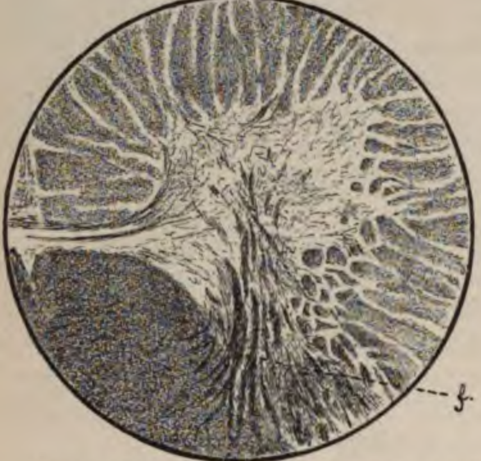


Cross-sections through the spinal cord in amyotrophic lateral sclerosis. The shaded portions are the diseased ones.

We find the same changes in the medulla and pons: *atrophy of the pyramidal tracts* and the motor nuclei of the *hypoglossus, facial, vago-*

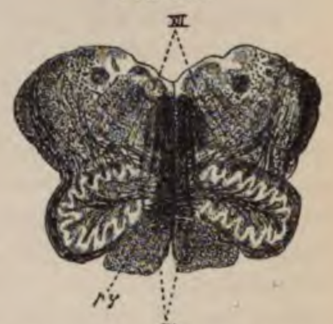
accessorius, and of the *motor fifth*. (Figs. 107, 108.) The degeneration of the pyramidal tract can be followed to the *cerebral peduncle* (Fig. 109), and in isolated cases has been traced to the internal capsule. Charcot and Marie, Mott, etc., found an atrophy of the pyramidal cells in the paracentral lobes. Nonne and Kaes observed a distortion of the fibræ propriæ and the projection fibres of the motor region of the cortex of the brain. There would then be a *disease of all the cortico-muscular conducting paths.*

FIG. 106.



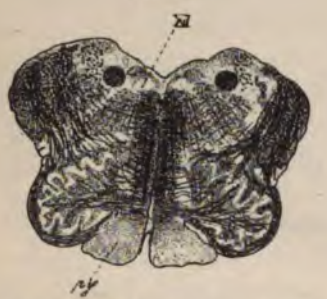
Atrophy of the anterior horns in amyotrophic lateral sclerosis, excepting the fibres coming from the posterior horn or the posterior roots (*f*). (Weigert's stain.)

FIG. 107.



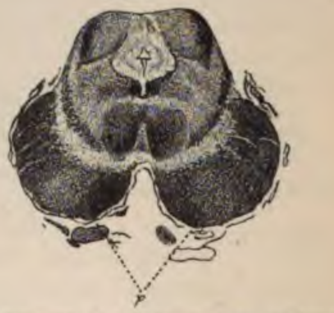
Normal medulla oblongata at the height of the hypoglossus. XII, hypoglossus nucleus; *w*, hypoglossus roots; *py*, pyramids. (Weigert's stain.)

FIG. 108.



Atrophy of the twelfth nucleus and its roots, as also of the pyramidal tract, from amyotrophic lateral sclerosis. (Weigert's stain.) (Compare with Fig. 107.)

FIG. 109.



Degeneration of the pyramid in cerebral peduncle from amyotrophic lateral sclerosis. (Weigert's stain.)

Differential Diagnosis.—Only typical cases can be differentiated from anterior chronic poliomyelitis. Chronic cervical myelitis may produce similar symptoms of atrophic paralysis in the arms and of spastic paralysis in the legs; but in it bladder and sensory disorders are generally present; the atrophy also does not extend to the lower extremities.

Cervical gliosis, if the posterior horns are not involved, may produce a clinical picture similar to amyotrophic lateral sclerosis, though this is rare, and, at least in the later stages, there occur symptoms referable to disease of the posterior gray matter. The disease also progresses more slowly.

Multiple sclerosis rarely evokes so marked a degenerative atrophy, and also presents characteristic cranial symptoms (nystagmus, optic atrophy, etc.).

Syphilitic processes have also occasionally produced a similar picture. Caries of the cervical vertebræ is generally accompanied by disorders of sensation and of the bladder; the simple atrophy accompanying chronic joint-diseases and accompanied by increase of the knee reflexes may make one think of amyotrophic lateral sclerosis.

In those cases in which the medulla becomes involved we do not find any difference between it and progressive bulbar paralysis (which see).

The prognosis as regards life is bad.

Treatment.—Endeavor to prevent the spasms by the methods already indicated; also apply galvanic currents to the medulla and spinal cord. In those cases in which there is trouble in swallowing, apply one electrode of the constant current to the nucha, and apply a labile current by means of the other to the region of the neck.

Fresh air and good nourishment are, of course, essential. Gowers recommends injections of strychnine.

PROGRESSIVE MUSCULAR ATROPHY.

The conditions grouped under this name are many, but all types are only varieties of the same disease. Two forms may, however, in general be considered as separate entities. First, *muscular atrophy or progressive spinal amyotrophy*; second, *primary progressive myopathy*. Even these two forms are often not sharply separated, transition forms connecting them.

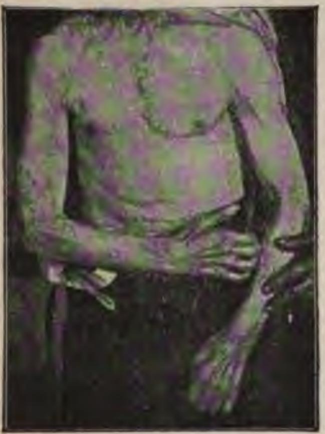
THE SPINAL FORM OF PROGRESSIVE MULTIPLE ATROPHY; SPINAL PROGRESSIVE AMYOTROPHY (DUCHENNE-ARAN).

This disease commences in middle life, rarely occurring before the twentieth year, though there is a family type which commences in early childhood. Excluding this type, heredity is not much of an etiological factor. Bernhardt has described an hereditary form occurring in adult life. Trauma and cold have been given as causes, as in every other nervous disease. In many cases over-exertion preceded the development of the disease, though there is a form of occupation atrophy which has nothing in common with progressive muscular atrophy. Males are affected more often than females. It is in general a rare disease. Duchenne overrated its frequency, as in his time gliosis and amyotrophic lateral sclerosis were included under it. Marie, however, goes too far in denying altogether the existence of this disease.

Symptoms.—Developing insidiously, it may last months, even a year, before noticeable disorders of function exist.

The small muscles of the hand are first affected. The opponens pollicis and the first interosseous atrophy first. The ball of the thumb flattens itself more and more, the interosseous space becomes depressed. The other small muscles of the hand follow, the muscular functions decreasing to a degree corresponding to the emaciation.

Fig. 110.



Localization of the atrophy in the spinal form of progressive muscular atrophy.

The deepening of the *interosseous spaces*, the *flattening* of the ball of the thumb and of the little finger, the *claw-hand* (Fig. 110), and the position of the thumb (in line with the fingers (ape-hand) or abducted and hyperextended) are the first objective signs noticed. Paralysis only results from atrophy. The weakness observed is merely a result of muscular decrease. The areolar tissue often atrophies also.

The atrophy is always accompanied by fibrillary twitching. We never find hypertrophy of the muscles.

An electrical examination shows a decrease of excitability conformable to the muscular atrophy; on the other hand, in some muscles and muscular groups we find a partial reaction of degeneration.

These phenomena occur in both hands, though occasionally one is affected more than the other.

There is no pain, or very slight pain. If paresthesia occurs, it is not a prominent symptom. Objective symptoms of sensory disturbance are not found.

The atrophy progresses slowly, but not by continuity. For example, it may spring from the hand-muscles to the shoulder, especially the deltoid. It may develop simultaneously in different groups, or may commence in the extensors of the lower arm, or in the muscles of the shoulder and back. But in all cases most of the arm, shoulder, and back becomes atrophied within a few years.

The healthy muscles gradually assume many of the functions of the paralyzed muscles. As the atrophy progresses, grooves and prominences appear, the contour of the bony structures becomes strongly evident, and the arm hangs helpless along the side.

The lower limb rarely becomes atrophied, or if so, very late in life. Long remissions may occur in the course of the trouble. Complete stoppage of the process or recovery does not occur. Where they have been observed, some other malady must have been present. The disease was once observed in combination with scleroderma. When the respiratory muscles become involved, or when symptoms of bulbar paralysis occur, life is in danger.

Differential Diagnosis.—Chronic anterior poliomyelitis develops more rapidly; a number of muscles are involved at the onset, even a whole limb; and paralysis occurs first, the atrophy following it. The paralysis often starts in the lower limb or in the shoulder region, and alterations in electrical excitability are observable before there is any noticeable atrophy. There are, however, transitional forms in which it is difficult to make a diagnosis.

In amyotrophic lateral sclerosis we find a spastic condition. In those rare cases in which this is absent the paralysis is very marked and may occur in muscles which are not atrophied. Some authors insist upon calling chronic anterior poliomyelitis, progressive muscular atrophy, and amyotrophic lateral sclerosis one disease.

Gliosis, pachymeningitis cervicalis hypertrophica, as well as caries of the lower cervical vertebræ, may also cause confusion in diagnosis. Gliosis is characterized by disorder of sensibility and trophic disturbances of the skin; the atrophy is also less symmetrical and confines itself longer, perhaps altogether, to one extremity. Pachymeningitis cervicalis hypertrophica commences with objective sensory disturbance in the region of the ulnar and median nerves, and with radiating pains. In the later stages an error is not possible.

Caries of the lower cervical vertebræ may produce muscular atrophy of the hands which simulates progressive muscular atrophy. Sensory disturbances and hyperesthesia of the spine are, however, generally present; if a gibbus is present, diagnosis is certain. The use of the X-rays may aid in making the diagnosis. Symptoms of compression myelitis also soon appear in caries, particularly spastic paresis of the legs, disturbance of the bladder, etc.

The occupation neuroses or professional pareses, however, are more liable to be confused with progressive muscular atrophy. The atrophy occurring in these is generally *unilateral;* also slight paresthesia and hypesthesia occur in the corresponding nerve-areas. The differentiation of these two diseases is important, because of the difference in prognosis. If, in doubtful cases, after the cause is removed, the process steadily progresses, we are justified in making a diagnosis of progressive muscular atrophy.

Arthritic muscular atrophy accompanies acute and chronic inflammations of the joints; it attacks the quadriceps when the knee-joint is diseased, the glutei when the hip-joint is affected, and the triceps and deltoid in disease of the elbow- or shoulder-joints, etc. It does not, as a rule, extend to the whole limb. The emaciation may develop rapidly. It is, however, a simple one, and is characterized by decrease, never by qualitative alterations, of the electrical excitability. When the joint becomes well, the muscular atrophy tends to disappear.

Some hold—and it has been made probable through experimental studies of Charcot and Krause—that the joint affection, by reflex action upon the anterior horns, may produce this atrophy; though this has been denied and the emaciation referred to mere inactivity (Strasser, Sulzer). Rarely does an acute inflammation of a joint spread directly to the adjacent nerves.

A diagnosis of progressive muscular atrophy should never be made from the *fibrillary tremor* alone. This may occur in normal persons after excess of any kind, in emaciated persons under the influence of cold, and especially in neurasthenic and hypochondriacal individuals.

Pathology.—The disease is a spinal one, an alteration of the *anterior gray columns* being always found, which consists principally in an atrophy of the cells and fibres. At the same time slight degeneration of the white matter of the anterior lateral tracts, secondary atrophy of isolated fibres in the vicinity of the gray matter, or a degeneration of the pyramidal tracts may be present. Although the combined degeneration of anterior horns and pyramidal tracts forms an anatomical basis for amyotrophic lateral sclerosis, this condition may be found in pure cases of progressive muscular atrophy (without spastic phenomena).

The *anterior roots,* the *muscles,* and their *nerves* take part in the atrophy. The muscles are emaciated, appear pale, yellowish-red, or yellow, and contain fatty stripes. Histologically we find decrease in size of the fibres, with degeneration of the muscular tissue to a fatty and granular matter. After the absorption of this, only the sarcolemma cylinders remain, filled with nuclei.

Treatment.—Of little avail. Subcutaneous administration of from one-half to one and a half milligram of strychnine once a day has been lauded by Gowers. Arsenic and thyroid extracts may also be tried. Exercise should not be allowed,—the muscles need rest. Smoking and drinking are also injurious. Mild electrical currents should be used. Climatic cures are futile.

HEREDITARY OR FAMILIAL TYPE OF PROGRESSIVE MUSCULAR ATROPHY OF SPINAL ORIGIN.

Werdnig and Hoffmann have observed cases of this kind which commenced in childhood and affected more than one person in the same family. The disease begins during the second year of life, in a subacute or chronic form, and causes weakness and atrophy of the muscles of the legs and back, which gradually invade the other muscles of the body. It progresses slowly and causes death in from one to six years. Werdnig has also observed bulbar symptoms.

PRIMARY MYOPATHY, DYSTROPHIA MUSCULORUM PROGRESSIVA.

Erb combined the former separate types of *pseudohypertrophy*, the *juvenile*, the *hereditary* (Leyden), and the *infantile* (Duchenne), under the title *dystrophia musculorum progressiva*, and showed that clinically and anatomically they are the same.

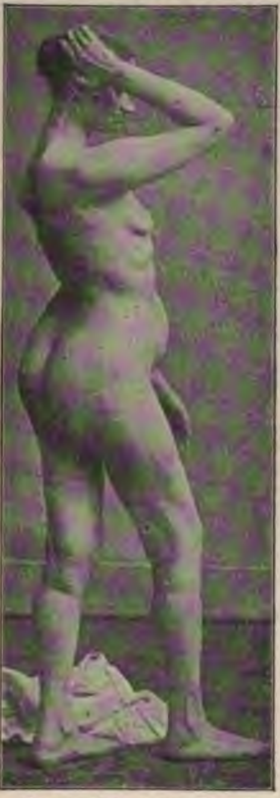
Primary myopathy presents the following differences from progressive spinal amyotrophy:

1. Onset in *early years.*
2. *Hereditary* character in most cases.
3. Commences in the *muscles of the trunk* and neighboring parts of the limbs, especially in the *pelvic girdle* and the muscles of the *lumbar* region, as well as in those of the thigh or shoulder and upper arm.
4. Combination of the atrophy with *true hypertrophy* and *pseudohypertrophy.*
5. Absence of fibrillary twitchings.
6. Simple *quantitative decrease* in muscular excitability, never reaction of degeneration.

Clinical Picture.—The disease commences in the first or second decennium, but may occasionally commence later in life. Several members of the same family are generally afflicted, or, it is transmitted from one generation to another, particularly through the mother. It develops slowly and is unnoticed for a long time. In those cases in which the muscles of the pelvis and thigh, as well as the extensors of the back, are involved, the first things that are noticed are a disturbance of gait and some difficulty in straightening the back.

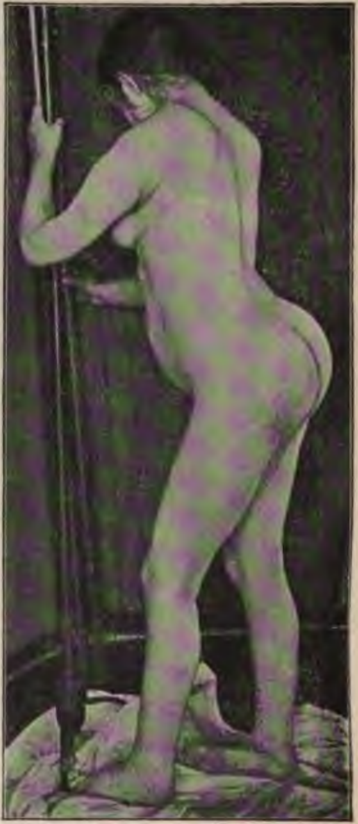
The gait becomes *waddling*, the pelvis, in walking, moves up and down more than usual, the climbing of stairs becomes difficult, and the patient falls easily. In rising from a sitting position, the patient supports himself by his hands, placing them on his thigh or knee, and by this help brings himself to an erect position. The manner in which he gets up from a recumbent position is characteristic. If he lies upon his back, he turns over on his stomach, and, keeping his hands on the floor, raises his pelvis so that he reaches a kneeling position; the knees are then raised so that only the hands and feet touch the floor; one hand is then placed upon the knee of the same side, and with a push the patient lifts himself, or he climbs up on his own legs by gradually supporting his hands on a higher and higher point of the thigh. A time, however, comes when the patient cannot raise himself any longer from the floor.

FIG. 111.



Progressive muscular dystrophy. Lordosis of the lumbar vertebræ.

FIG. 112.

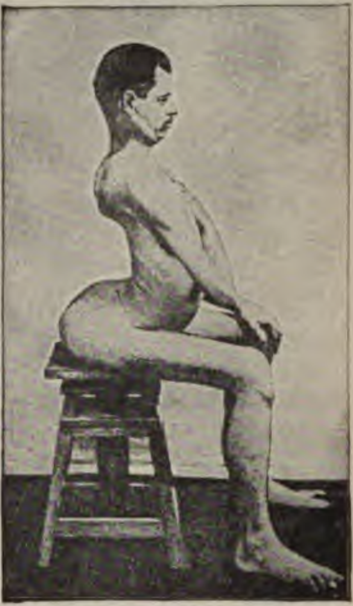


Progressive muscular dystrophy. Lordosis from atrophy of the abdominal muscles, etc.

Another anomaly that occurs is *lordosis* of the *lumbar* vertebral column (Figs. 111 and 112). The abdomen is protruded and the shoulders are thrown back. Paralysis of the abdominal muscles may also produce lordosis. Although it generally disappears when the patient sits down, it may occasionally be present even then. (See Fig. 113.)

Resulting from the paralysis of the *trapezius*, *pectoralis major*, *latissimus dorsi*, and *serratus anticus major*, the shoulder-blade becomes abnormally movable and follows every movement of the upper arm. Weakness of the shoulder-muscles causes an abnormal position of the shoulder-blade and disturbances of motility which are especially noticeable in elevating the arms. If we attempt to lift the patient, placing our hands in the axillæ, the shoulders, and not the body, are raised.

FIG. 113.



Advanced case of progressive myopathy. Pronounced lordosis in sitting. (After Souques-Brissaud.)

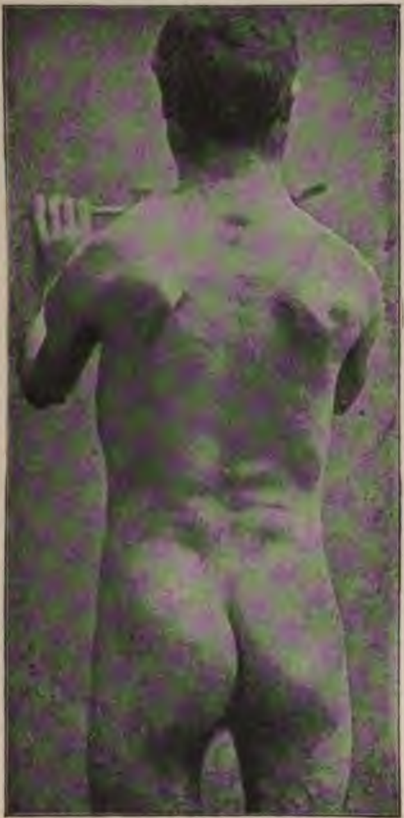
In rest the shoulders are depressed downward and forward; the acromion stands lower than the inner upper border; the shoulder-blades stand wide apart and wing-like from the thorax. In raising the arm the characteristic position observed in trapezius paralysis is produced. (Figs. 114 and 115.)

Close examination shows that the following muscles are, as a rule, involved: Trapezius (the upper portion is often spared), serratus anticus major, the sternocostal portion of the pectoralis major, the latissimus dorsi, rhomboidei, infraspinatus, deltoid, biceps, brachialis internus and supinator longus, the erector trunci, the glutei, quadriceps, adductors, and the muscles of the calf, and a part of the peroneal region. The distal parts of the extremities are rarely involved.

Atrophy with noticeable loss of substance occurs in only a part of the muscular system; another part shows *increase in volume* due to an *hypertrophy* of the *areolar* and *connective tissues*, in part also to a *true hypertrophy* of the muscular fibres. The contour of the muscles is thereby altered. Some are flattened, others increased in volume, and, when the hypertrophy confines itself to a single part, an intumescent appearance is given to the region.

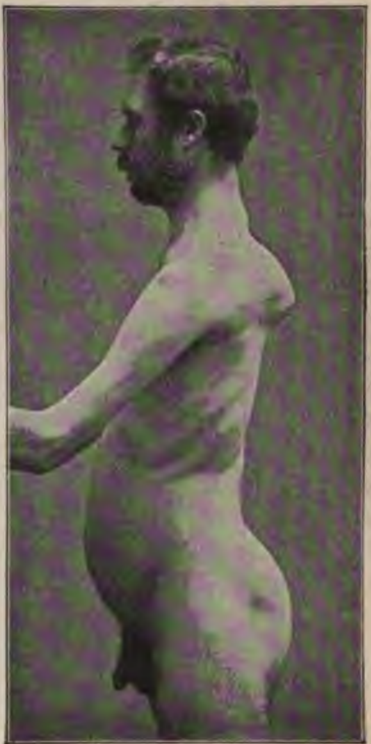
The atrophy attacks particularly the pectoralis major, trapezius, serratus anticus major, latissimus dorsi, biceps, brachialis internus, quadriceps femoris, adductors, etc., while the hypertrophy seems to seek out especially the infraspinatus, deltoid, triceps, sartorius, glutei, and, above all, the calf-muscles.

FIG. 114.



Progressive muscular dystrophy. Juvenile form. Position of the shoulder-blades from atrophy of the trapezius and serratus anticus major.

FIG. 115.



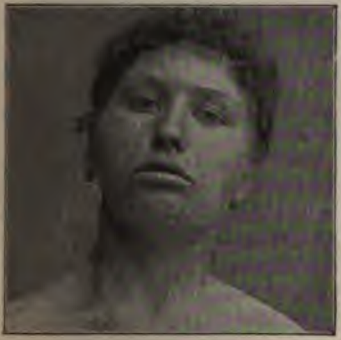
Same as Fig. 114. Lateral view.

The *orbicularis oris* and *palpebrarum* are often involved. (Figs. 116 and 2.) In advanced cases the face may become mask-like (facies myopathica).

Both sides of the body are involved, but not always symmetrically; the muscles react to electrical stimulation, though the excitability is lowered, the mechanical excitability being also decreased. The deep reflexes are normal or decreased, and the sensibility is normal. No bulbar or bladder symptoms are found.

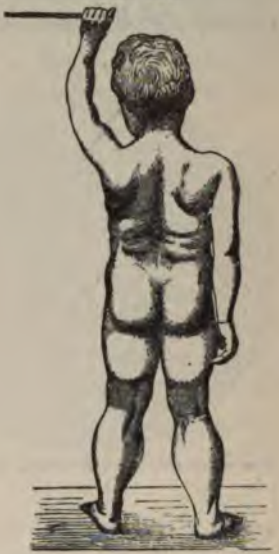
The disease is chronic. I observed a woman who, though fifty-eight years old and sick with this disease since early childhood, until a few years ago was able to move around slowly. On account of its insidious nature, the normal muscles learn to take upon themselves the functions of the diseased muscles; and one is often surprised at the command which the patient, notwithstanding his muscular weakness, has over his movements. Occasionally paralytic contractures may develop, as in the triceps suræ, biceps, and flexors of the knee.

FIG. 116.



Myopathic facies. Patient is unable to purse the puffed-up (pseudohypertrophic) lips.

FIG. 117.



Progressive muscular dystrophy. Pseudohypertrophy. (After Erb.)

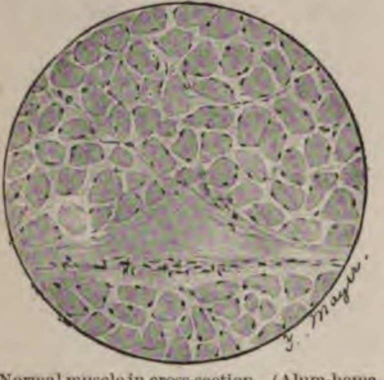
Varieties.—The *juvenile* form develops in youth and middle age; the shoulder and upper arm are first and chiefly involved, and the true and false hypertrophy is confined to a few muscles. The pseudohypertrophy commences in childhood, in males more often than in females, and involves the lower half of the body more than the upper, the atrophy appearing particularly in the upper. (Fig. 117.)

The *infantile* form is characterized by the primary involvement of the facial muscles. The *hereditary* form develops from the eighth to the tenth year, or later, commences with weakness of the back and lower extremities, and is purely hereditary.

Pathology.—The spinal cord and peripheral nerves are generally normal. We find the following changes in the muscles: Atrophy and hypertrophy of the primitive fibres associated with each other, increase of the muscle nuclei, proliferation of the internal perimysium with fatty deposits in it, splitting and longitudinal striation of the muscular fibres, and formation of vacuoles in them. Figs. 119, 120, and 121 show the most important changes. The anatomical process does not, however, form a sure basis for differentiation of the different forms of progressive muscular atrophy (Cramer). Erb believes that hypertrophy of the fibres precedes the atrophy. There is no doubt that congenital developmental anomalies of the muscular system are the cause of this disease.

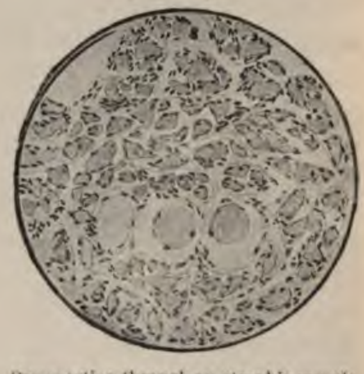
Differential Diagnosis.—In advanced cases no error in diagnosis can be made. Difficulty is experienced only in those cases in which atrophy and hypertrophy counteract each other so that there is no alteration of muscular volume. Even in these cases, however, the alteration in electrical excitability and the disturbances of function, as well as a peculiar rounding of the limbs, sausage-like, show clearly what the disease is.

FIG. 118.



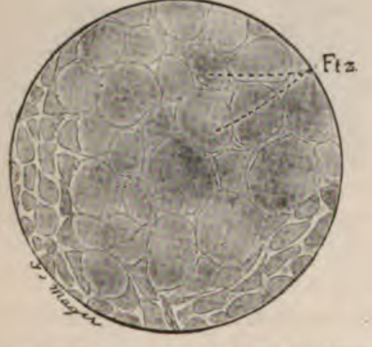
Normal muscle in cross-section. (Alum-hematoxylon.)

FIG. 119.



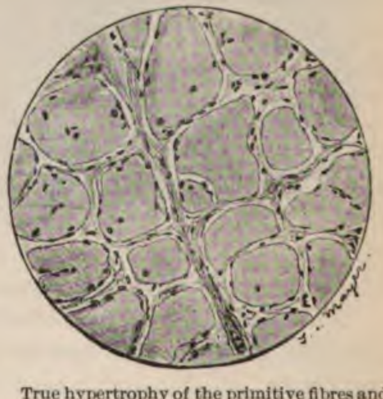
Cross-section through an atrophic muscle.

FIG. 120.



Progressive muscular dystrophy. Cross-section through a lipomatous-degenerated muscle. *Ftz*, fat-cells.

FIG. 121.



True hypertrophy of the primitive fibres and dilatation of the internal perimysium from progressive muscular dystrophy. Cross-section.

In inflammatory processes of the vertebræ or muscles of the back, the pain, sensitiveness to touch, elevation of temperature, and objective evidence of some vertebral trouble will prevent error in diagnosis. I saw a case of acute lumbar poliomyelitis in which the inability to extend the vertebral column simulated incipient dystrophy. In another case of post-diphtheritic paralysis, which involved the muscles of the lower part of the back, the lordosis, as well as the manner in which the child raised himself from a recumbent posture,[1] resembled the type described above. The microscopical examination made in my laboratory by Dr. Sano confirmed the diagnosis.

Congenital muscular defects confine themselves to a few muscles, and are not progressive.

Complications.—This disease may be combined with hysteria, epilepsy, and idiocy. I have seen it once combined with tabes, also with poliomyelitis (Cassirer). Bernhardt described it in combination with periodic paralysis of the extremities.

Prognosis.—Life is not endangered except when the diaphragm and the respiratory muscles become involved in the dystrophy. Those affected do not, however, in general, reach an old age. The earlier the trouble commences, the earlier does death result. The juvenile form has therefore a better prognosis than the others. Some cases, however, live a long time, and in others the disease does not progress after a certain stage. Abortive forms also occur.

Treatment.—Fresh air, nourishing diet, with exclusion of fat-forming foods, moderate exercise, hydropathic and electrical treatment (galvanic and faradic) are all in place. Drugs are of no avail. Tenotomy has been done in some cases, with shortening of the Achilles tendon, with good results.

THE SO-CALLED NEURAL FORM OF PROGRESSIVE MUSCULAR ATROPHY. (PERONEAL TYPE OF PROGRESSIVE MUSCULAR ATROPHY. TYPE OF CHARCOT-MARIE, TOOTH.)

The disease commences, as a rule, in the second half of childhood, but may occur even in the third and fourth decennium. Almost always several members of a family are attacked. It can be directly inherited (through the father), or may skip a generation. Men are affected more than women.

The development is slow. The atrophy commences, in the greater number of cases, in the muscles of the foot, especially the peroneal muscles, the extensor communis digitorum, and the small foot-muscles. A pes varus, equinus, or equino-varus is the result. The ankle-joint becomes ankylosed; it may, however, be loosened. The lower leg is emaciated. Later the muscles of the calf of the leg become atrophied.

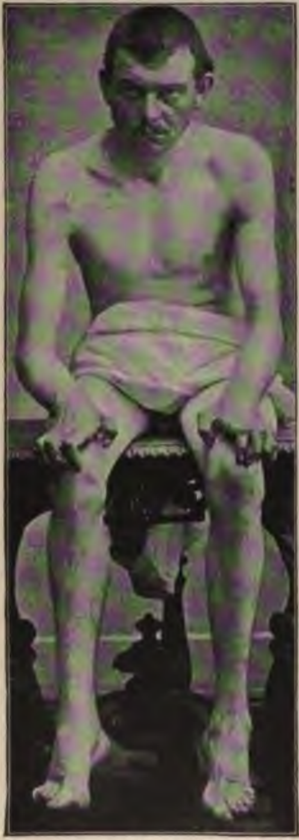
---

[1] I have observed in the traumatic neuroses also, after contusion of the spinal region, the same manner of "arising upon oneself."

After some years the upper extremities become involved, the thenar, hypothenar, and interossei becoming atrophied first. A claw-hand results. Fibrillary twitchings take place. Electrical examination shows an incomplete reaction of degeneration, also sluggish reactions to the faradic current. The unparalyzed muscles may also show electrical changes. I saw one case in which severe disturbance of the electrical excitability extended over almost the entire body, while the muscular atrophy only existed in the lower extremity. The nerves are for the most part not particularly sensitive to touch.

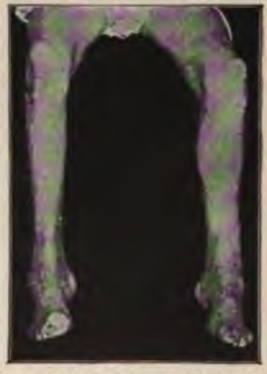
The knee reflexes are abolished. *Sensory disorders* may occur,—pain and decreased sensibility in the distal parts of the extremities. I observed a *mal peforans* in one case. All other functions are undisturbed. It runs a chronic course; remissions may occur, or the disease may cease progressing altogether. A high old age may be reached. Surgical orthopedic procedures (tenotomy etc.) may produce good results.

FIG. 122.



Case of peroneal type of progressive muscular atrophy.

FIG. 123.



The legs and feet in a case of the peroneal type of progressive muscular atrophy.

Thyroidin is said to have been of benefit in one case (?).

We know little concerning its pathology. In the observations of Virchow and Friedreich, alterations in the peripheral nerves and in Goll's tracts were found. Hoffmann believed it to be a chronic hereditary form of multiple neuritis; hence the name neurotic or neural type of progressive muscular atrophy. Dubreuilh's and Marinesco's observations seem to confirm this. I have been able, with the co-operation of Dr. Cassirer, to prove that the clinical picture can result from a primary myopathy (for which I propose the name of myositic form of progressive muscular atrophy).

Fig. 122 illustrates a case in which the hands were more involved than the lower limbs. In Fig. 123 we have a clearer picture of the changes in the lower limbs.

Dejerine has lately described some cases which are allied to the above diseases, but which take us farther away from the type of progressive muscular atrophy. He describes them thus: The atrophy begins in the distal parts of the extremities in childhood, progresses gradually with severe lightning pains, sensory disturbance, ataxia, myosis, sluggish pupils, nystagmus, Romberg's symptom, and kyphoscoliosis. No reaction of degeneration; quantitative decrease in electrical excitability; marked hypertrophy and induration of the nerves. Pathological examination: Chronic interstitial neuritis decreasing from periphery to centre, also of the posterior roots, and sclerosis of Goll's and Burdach's columns. Strümpell believes that Dejerine's cases are a combination of Friedreich's disease with the hereditary peroneal type of progressive muscular atrophy.

THOMSEN'S DISEASE (MYOTONIA CONGENITA).

This is a disease which attacks *several members of the same family* and which descends from one *generation* to the next. More than twenty cases in four generations were described in the family of Dr. Thomsen, to whom we owe the first exact description.

It generally manifests itself in *early childhood*, though it may not appear until puberty, or later. Those individuals affected possess a well-developed, perhaps a *hypervoluminous* musculature. The muscular strength may, however, be decreased. The most important phenomenon noticed in childhood is the inhibition of voluntary movements due to a *muscular rigidity* which sets in during their execution. In attempts to use a muscle group after a long rest, a condition of tonic spasm ensues, and the will is not able to relax the muscles. After five, twenty, or thirty seconds the spasm relaxes, and then each repetition of the movement induces a slighter spasm until none is noticeable any longer.

The *myotonic* disturbances are especially noticeable when the individual suddenly attempts a forcible movement, as suddenly and forcibly clenching his hand or flexing the lower arm. The hand extended to grasp a glass may remain rigidly stretched; the hand in clasping another's hand may hold it as in a vice until the contraction disappears. In some cases after any movement the patient falls to the floor and lies there as rigid as a stick until the muscles again relax.

Although the whole muscular mass is generally affected, it occasionally happens that only a certain region or a definite muscular group is affected.

Myotonia is increased through the influence of the feelings, or by long rest, forced exertion, fever, cold, or when the patient believes himself *observed*. Warmth, mental rest, frequent repetition of movements, and the moderate ingestion of alcohol have a favorable influence.

The objective examination shows the following phenomena of the muscular apparatus:

1. *Increase of mechanical muscular irritability. Muscular percussion produces a sluggish tonic contraction of the part struck, the contraction lasting some time.* Pressure with the finger-tip is often sufficient to produce it.

2. *Alteration of electrical irritability, the myotonic* reaction. The reaction of the nerves to faradic currents is about normal. Stimulation of the nerves with stronger currents produces a tonic muscular contraction of *long duration;* single applications of the open induction current produce short twitchings. The direct faradic muscle excitability is increased. Weak currents even produce a tonic contraction of long duration. With a continued faradic stimulation an undulation of the stimulated muscle is often noticed. *Galvanic excitability* of the nerves is somewhat lowered. Only labile currents produce a *continued* contraction. *Direct* galvanic excitability of the muscles is somewhat increased; only closure contractions are evoked; generally the *ACC* is in the ascendency. Especially noticeable, however, is the *sluggish, tonic character* of the *muscular contractions* and their *long duration.* A stabile galvanic current produces a *rhythmical* undulation of the muscles, a contraction-wave passing from the cathode to the anode. To produce this phenomenon strong currents (to twenty milliamperes) are necessary.

Static sparks produced only single contractions in a case examined by me.

This is all that is found. The other functions of the nervous system are not involved. Complications with *psychical disorders, epilepsy, hemicrania*, etc., are not rarely observed. Combinations with pseudohypertrophy (Charcot), multiple neuritis (Hoffmann), and tetany (Bethmann) have been described. It is found in all degrees of severity.

The diagnosis is not difficult. Eulenberg has described a similar disease as *paramyotonia congenita.* This disease results from cold, which causes a numbness and stiffness. After this rigidity has passed away a kind of paralytic weakness ensues. The orbicularis oris and palpebrarum were especially involved. The mechanical muscular irritability was not increased. Some cases have been described which seem to be a combination of these two diseases.

The myotonia acquisita of Talma, the ataxic paramyotonia of Gowers, etc., have little in common with Thomsen's disease.

The intentional spasms of tetany (Kasparek) are very like those of myotonia, but the other criteria of this disease make a diagnosis certain. Hoffmann found the myotonic reaction in a case of strumiprivous tetany with myxedema.

Prognosis is good as to life; but no improvement can be expected. It lasts a lifetime without any progression, but with frequent remissions.

Pathology.—Erb and others have found a *hypertrophy* of the primitive fibres, an increase of the sarcolemmar nuclei, and a slight increase of the interstitial tissue. Jacoby found the sarcode muscular elements increased, lessened in size, and lying closer together. Dejerine and Sottas found in a case the same changes without any lesion of the central nervous system. Similar phenomena have been produced in animals by poisoning with creatinin. But the theory that it is due to auto-intoxication has no foundation, while everything indicates that it results from an abnormal development.

As I and Bechterew have claimed before, rational gymnastics is the only method of treatment that is of benefit. Massage may also be tried.

THE DIFFUSE DISEASES OF THE SPINAL CORD.

Most of the diseases under this head originate in the spinal membranes, or in the vertebræ themselves, or from distant organs. Aortic aneurisms and malignant tumors starting from the kidneys or from the retroperitoneal lymph glands may break through and involve the spinal cord. Decubitus also extending inward may cause suppuration of the sacral canal and infect the meninges and spinal cord.

Other forms arise in the spinal cord directly, and, although they may be produced by infectious processes in other parts of the body, pathologically they are primary and independent diseases of the spinal cord.

A. DISEASES OF THE SPINAL CORD CAUSED BY VERTEBRAL DISEASE.

Injuries, fractures, and luxations will be only slightly touched upon, as a more complete description belongs to a text-book of surgery.

DISLOCATIONS AND FRACTURES OF THE VERTEBRAL COLUMN.

Dislocations occur most often in the region of the cervical vertebræ, and especially between the fifth and sixth and between the first two cervical vertebræ. They rarely happen in the dorsal or lumbar region.

It is generally bilateral (flexion or total dislocation), more rarely unilateral or an abduction dislocation, and may be complete or incomplete. The upper vertebra, called the luxated one, is almost always pushed forward. The dislocation is generally a result of indirect force, excessive flexion of the head and neck, or falls and blows on the head.

Deformity is the most important direct symptom. In bilateral dislocations of the cervical vertebræ the head is inclined forward, the spinous process of the vertebra below the luxated one is very prominent, while the former is inclined forward. An abnormal prominence may be detected in emaciated persons on the side of the neck, and, when the upper cervical vertebræ are involved, in the pharynx by palpation. The spinal column is held rigid by the contracted muscles; every attempt at movement is painful.

In unilateral dislocations the head is inclined towards the opposite shoulder, the chin, however, being turned towards the same side.

Vertebral fractures occur in every region, but especially in the region of the middle cervical, the upper dorsal, and the first lumbar vertebræ. It generally results from some severe external force, as falling on the head or buttocks, or some heavy weight falling upon the patient's head. Forcible muscular exertion may occasionally cause vertebral fractures. Dislocation and fracture are often combined. The symptoms of dislocation are very similar to those of fracture. Marked dislocation of a vertebra with fixation of the part above denotes the first. Crepitation is found only with fractures. More rarely fragments may be palpated.

What symptoms arise on the part of the nervous system? There may be none, especially in partial injuries. As a rule, however, we have some contusion, laceration, or pressure of the cord, with corresponding symptoms. The cord may be injured, even though there is no separation in continuity of the vertebræ. *Hemorrhage* into the *epidural space*, *subarachnoidal hemorrhages*, and *central hematomyelia* may be produced thus.

Injuries to the first two cervical vertebræ generally cause instant death. We have dislocation, inclination, and turning of the head, rigidity of the back of the neck, and severe local pains, and *radiating pains* in the region of the *upper cervical nerves*, especially the great occipital. Dyspnœa, and often even bulbar symptoms, or paralysis of the muscles innervated by the cervical nerves, may be present. If death does not immediately occur, a fatal termination is generally reached some time after, through a careless movement of the head or from a myelitis of the upper cervical segment or of the medulla oblongata.

A common symptom of compression or laceration of the cord, especially of the cervical region, is the occurrence of *erections of the penis.*

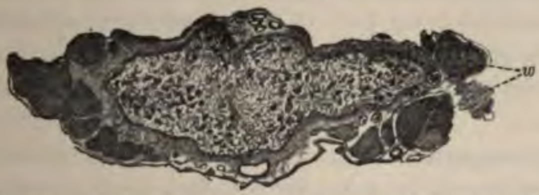
Ejaculation may occur at the moment of the injury; under these conditions the erection lasts some time. *Considerable elevation of temperature, decreased or increased pulse-rate*, and *vasomotor disturbances* are also noticed.

In injuries of the third and fourth cervical vertebræ life is mainly in danger from involvement of the *phrenic nerve.*

In general the following rules may be established concerning spinal symptoms accompanying fractures and luxations.

The cord is generally so altered in the region of the injury, through compression, laceration, hemorrhage, or inflammation, that conduction is more or less lost. All those muscles whose motor paths lie in the segment below the lesion are paralyzed,—*i.e.*, in diseases of the upper cervical cord, the muscles of all four extremities and the trunk; in diseases of the dorsal cord, the legs and a part of the trunk are affected. Also all sensation is lost in those zones of the skin whose sensory nerves enter a segment of the cord below the lesion. In most cases the upper border of the sensory disturbance does not reach as high as the affected vertebral segment. Consult the chapter on Localization for more complete particulars.

FIG. 124.



The spinal cord in a case of fracture of the vertebral column. Complete destruction of the white matter and adhesion of the meninges to each other and to the roots (*w*).

Inasmuch as in compressions of the cord and roots the latter often remain uninvolved, while the more delicate cord degenerates, the paralysis and anesthesia need only extend upward to that root-zone which arises from the corresponding segment of the cord; for example, in injuries of the fourth dorsal vertebra, to the region of the sixth intercostal nerves. If the roots as well as the cord are involved, the paralyzed part extends upward to the corresponding root-zone. Often not the vertebra whose spinous process appears prominent dorsally, but the one above or below produces the compression.

When an incongruity exists between the seat of a vertebral lesion and the extent of the paralysis, rest assured that the cord is only partly affected. It sometimes results from a central hemorrhage; compression of the cord may injure the soft, vascular gray tissue, while the white substance remains intact. The sensory roots which enter here are not therefore involved, and the sensory conduction is unimpaired. This contradicts Flatau's experiments.

A hyperesthetic zone or herpes zoster is often found on the upper border of the anesthetic region. In these transverse lesions, wherever their seat may be, the *function of the sphincters* is impaired. Retention of the urine with incontinence or incontinence alvi and decubitus occur early or later. Partial lesions may not present these symptoms.

It is also the rule that in severe spinal-cord lesions, with total loss of conduction, the paralysis of the lower limbs is a flaccid one, with loss of all reflexes. This is certain for some time following the injury when the shock has involved the whole cord.

Bastian has lately set up the hypothesis that diseases or injuries which produce a complete destruction of conduction in the cord cause a total loss of all reflexes of the lower segment of the cord.

In a transverse lesion of the cervical cord the plantar and knee reflexes will be annulled, with complete paralysis and anesthesia. Thorburn claims that the reflex functions of the bladder and rectum are also annulled, but this is disputed by Kocher.[1] This also contradicts most of the earlier observations.

It was formerly an axiom that a disease of the cervical or dorsal cord which involved every part of a cross-section produced spastic contraction of the paralyzed muscles and increased reflexes. Bastian explains this by saying that the observations were mostly of partial breaks in conduction.

Bruns has supported Bastian in his hypothesis, both clinically and anatomically, although he acknowledges that the skin reflexes may be increased, so that, notwithstanding the atony, a sort of reflex spasm may be found in the legs.

In *partial* lesions of the spinal cord which have their seat above the lumbar prominence the tendon reflexes may be lost, but only at first; later a spastic condition with increased deep and skin reflexes ensues. When the sensibility is not completely lost, a partial break in conduction probably exists. Paresthesia, pain, and hyperesthesia of the lower limbs are also probably indicative of partial disturbance (in cervical or dorsal lesions). The same is true of normal or increased ability for urination and defecation.

If the sensibility only is affected, while the motor system is intact, the nerve-roots alone are involved. Compression of the nerve-roots of the cervical prominence, the lumbosacral cord, and the cauda equina produce marked symptoms. The involvement of the posterior roots produces radiating pains, which are felt in the peripheral nerves, and hyperesthesia and anesthesia. Involvement of the anterior roots evokes atrophic paralysis of the muscles innervated by them; tremor, twitchings, and contractures may also be the results of compression of the anterior roots. Injuries of the cervical prominence produce a total paralysis of the legs and trunk, while in the arms certain muscles are uninvolved. If, for example, the compression occurs below the origin of the fifth and sixth cervical roots, we find the deltoid, biceps, brachialis internus, and supinators free from all paralysis, while the forearm and hand-muscles are paralyzed. A tonic contraction may occur in the unaffected muscles, producing abduction, rotation outward, flexion of the lower arm, etc. (Thorburn.)

---

[1] This author shows also that the erection reflex remains. He also described a peculiar testicular reflex,—a contraction of the abdominal muscles (of the same side) produced by pressure of the testicle.

The lower in the cervical cord the seat of the injury is, the fewer the muscles that are involved.

An analogous relationship is found in sensory disorders.

Oculopupillary symptoms are especially liable to occur in disease of the first dorsal segment. Myosis and narrowing of the palpebral fissure are also symptoms of fractures and dislocations of the fourth to the sixth cervical vertebræ. Vasomotor changes are often found following dorsal injuries, as is also priapism.

Thorburn, to whom we owe much for his observations of these conditions, claims to have detected alterations of the back of the eye following upon traumatism of the cervical cord.

Contusion or compression of the lumbosacral segment (twelfth dorsal and first lumbar) produces deeper and more lasting injuries, not only of the cord, but also of the lumbar and sacral roots,—*e.g.*, we see the same phenomena resulting from fracture of the first lumbar vertebræ that are produced by injury to the conus terminale,—paralysis of the bladder, rectum, and of the genitals, with anesthesia in the region of the third and fourth sacral nerves, the lower limbs having normal movement.

If, however, the roots surrounding the conus are involved, we have also atrophic paralysis of the lower extremities and the knee reflexes are lost.

The plexus lumbalis and plexus sacralis are paralyzed when the tenth, eleventh, and twelfth dorsal vertebræ are dislocated or fractured. If the upper lumbar cord is not involved, the sensibility in the ileo-hypogastric and ileo-inguinal regions, etc., is normal, and the flexors and adductors of the thigh may be partly used. If, however, the roots surrounding the lower lumbar cord are also involved, all the parts are affected. When the third and fourth lumbar roots and the segment from which they arise are intact, the knee reflexes remain normal, as well as the sensibility in the region of the obturator and crural nerves, the motor disorder being confined to the sacral and coccygeal plexuses.

Fracture of the first lumbar vertebræ may involve the third and following sacral segments, the first two escaping. We have then the saddle-form type of anesthesia. Testicular sensation is unimpaired; coitus is impossible, although sexual desire and erections may be intact.

Injuries of the *cauda equina* are hardly distinguishable from those of the conus; the more so as the cauda is surrounded by the roots of the conus. Lesions of the cauda produce radiating pains in the region of the sciatic, pudendal, etc., nerves, which are generally absent in diseases confined to the conus. Paralytic symptoms in injuries of the cauda are generally less complete and less symmetrical. The prognosis is also a better one.

SPINAL CONCUSSION may produce symptoms of a severe spinal injury, which, however, soon disappear. Kocher declares that it is not functional; that a hemorrhage, traumatic necrosis (Schmaus), or other disorders of the spinal cord are present. Although this is true in many cases where a functional disorder was thought to be present, still we believe that there is such a condition as functional spinal concussion, though we must acknowledge that most of the symptoms are *cerebral* (see chapters on Traumatic Neuroses and Cerebral Concussion).

Vertebral injuries may also produce spondylitis or traumatic spondylomalacia (Henle). The X-rays are of much service in making a diagnosis of vertebral injuries.

The prognosis is a grave one, it being almost always fatal in cervical, less so in lumbar injuries. Dislocations do not have so bad a prognosis as fractures. When the bladder and rectal paralysis does not recede after the eighth or ninth week, Gurlts claims that recovery is not to be expected. The less the cord and roots are involved, the better is the prognosis. Recovery occurs quickly, within some days or weeks, or not at all. In those cases where death does not result, the bladder and rectal paralysis, the cystitis, and the decubitus form the chief dangers.

Treatment.—Great care must be taken in examining, transporting, and laying down the patient. Avoidance of every unnecessary manipulation of the wounded parts is the most important rule. He should be so placed that the injured part is well supported and fixed and that every active movement may be avoided.

Attempts to put a dislocated vertebra in place should be made only when symptoms of compression are present. Text-books of surgery give full details of the methods to be used. In fracture cases all efforts at reposition should be avoided.

To prevent decubitus, water-pillows should be used and the buttocks and the heels should be protected with rubber or cotton pads. Naturally, great cleanliness is necessary in catherization. Kocher recommends continuous drainage, using a Nélaton, and having it descend into a basin filled with some antiseptic fluid. In the first few days give opiates, to prevent any passage from the rectum.

Many attempts have been made to relieve the patient *operatively* by *laminectomy*, removal of fragments, etc. Of one hundred and sixty-seven cases operated upon, according to Chipault, only twelve have been relieved and twenty-four improved. Later results have, however, been somewhat better, as noticed by Schede.

Early operation is always contraindicated. Only in those cases where we are assured that only a *partial* break in conduction has occurred are we justified in operating. Do not operate before the sixth week nor later than three months after the accident.

Goldscheider draws the following conclusions:

1. Recent cases should not be operated upon, except where there is simply a comminutive fracture of the *vertebra*, fragments of which are pressing upon the cord.

2. When the paralysis does not disappear, and a deformity also exists, indicating a fracture of the vertebral body, an operation may be in place.

3. Hemorrhage into the spinal canal is not a sufficient cause for laminectomy.

4. Operation is more justifiable in fracture of the lower lumbar vertebræ than in any other region. All this is, however, hypothetical. We are still feeling our way in this question.

CARIES OF THE VERTEBRAL COLUMN; TUBERCULAR SPONDYLITIS.

Although generally tuberculous, caries may result from traumata and other causes. Tubercular osteitis of the vertebræ consists in the development of fungous granulation tissue, which produces liquefaction of the bones and formation of caseous and purulent matter. It generally affects only one vertebra, and may undergo resolution, new bone being produced to take the place of the degenerated. If it progresses to necrosis, or if the fungoid, caseous, purulent matter enters the spinal canal, the spinal cord may become involved.

Compression of the cord or sudden breaking down of the vertebral column may also, however, be the cause of the spinal-cord lesions. The fungoid masses or an abscess may press against the dura and produce a chronic external pachymeningitis. The compression and closing of the dural arteries and lymph-vessels can cause an *anemia*, and afterwards a *passive congestion* which may lead to *softening*. All these, of course, may terminate in an acute process, an interstitial transverse myelitis. In only a few cases, however, does a myelitis exist from the first. Tuberculosis may follow the course of the arteries and produce an arteritis obliterans, and thus softening (Schmaus).

The edema, softening, or myelitis is generally confined to a small segment, about one-half centimetre to one-half inch thick. Small lesions may also occur in other parts near by, and if of long standing, secondary degenerations may occur. We rarely, however, find disseminated myelitic foci. The spinal roots are also affected. The dorsal region is most often involved. This disease is due to tuberculosis or scrofula. The caries may, however, be the only manifestation of the constitutional disorder. Often other symptoms of tuberculosis are found (carious processes of other bones and joints, purulent glands, tuberculosis of the lungs, etc.). Children are most often attacked, it occurring either spontaneously or after some trauma, etc.

Symptomatology.—Fever is not necessarily present, nor any other general symptoms.

Most of the symptoms are a *direct result of the vertebral disease* and indicate some spinal trouble.

Pain is present in the region of the diseased vertebra, and is increased by pressure or movement, so that the patient avoids bending his back. In caries, pressure produces pain, especially deep pressure or percussion, and the sensitiveness is localized in one or two neighboring spinous processes. A sponge soaked in hot water or the cathode of a galvanic current stroked over the cord will produce pain as soon as the diseased vertebra is reached.

The most important symptom is the *deformity*,—the *acute-angled kyphosis*, or Pott's hump. A gravity *abscess* is a frequent symptom, the pus descending downward till it breaks through the skin or in cervical caries passing backward (retro-pharyngeal abscess). It may descend along the psoas and the large arteries to appear near Poupart's ligament or on the anterior part of the thigh.

Pain, girdle-sensation, sometimes hyperesthesia, but generally hypesthesia or anesthesia and herpes zoster, may all occur as indications of involvement of the cord and its roots.

If the *eighth cervical* and *first dorsal* roots are affected, we find pains and sensory disorders in the ulnar region, atrophic paralysis of the small hand-muscles, etc., and oculopupillary phenomena. If the fifth and sixth cervical roots are involved, we have paralysis, with atrophy of the *deltoid*, *biceps*, *brachial internus*, and *supinator longus*, and the anesthesia extends above the deltoid and on the outer part of the arm, etc.

The symptoms on the part of the spinal cord are variable according to the height at which the compression takes place. In the most common form, *caries dorsalis*, the symptoms are about the following:

1. *Paraplegia of the legs with rigidity* and increased tendon reflexes.
2. *Anesthesia* in the area innervated by the roots which arise in the diseased part of the cord. A hyperesthetic zone may be present immediately above the anesthetic or hypesthetic zone.
3. *Girdle-sensation.*
4. Skin reflexes are *present* or *increased* in the lower limbs.
5. *Disorders in bladder* or *rectal functions.*

Decubitus may also be found, as well as other trophic disorders. If, however, there is a complete break in connection (which is rarely the case), the reflexes and muscular tonicity are found in an opposite condition, according to Bastian. This has been discussed in previous chapters.

If the caries attacks the eleventh and twelfth dorsal vertebræ or the first lumbar, we have, in place of a spastic paralysis, a *flaccid* paralysis of the legs, with *atrophy*, and the reflexes are *weakened* or *lost*. Caries of the lower cervical region is differentiated from caries dorsalis by the fact that in it the spastic paralysis of the legs is accompanied by an atrophy of the upper extremity. The paralysis also extends to the thoracic and abdominal muscles and produces difficulty in respiration, especially weakened expiration, which may be fatal if bronchial catarrh is present. If the compression occurs above the cervical prominence, we find spastic paralysis in all four extremities, which may for a short time confine itself to the arms, more rarely to the legs. The sensory disorder covers a wide extent. If the *phrenic* nerve is involved, inspiratory dyspnœa occurs.

We have presupposed, in describing the spinal-cord symptoms, that the compression had caused a more or less complete break in continuity, and have ignored the ideas derived from Bastian's hypothesis. This is, however, not always the case.

It more often occurs that the break is a partial one, and then the symptoms are but incompletely developed. The disturbance of motility is then always more marked than that of sensation. The latter may be absent altogether. The most constant sign of cervical and dorsal caries is the *spastic paresis;* if it increases to paralysis, the other symptoms, the anesthesia and the paralysis of the sphincters, appear also. Brown-Séquard's symptom-complex rarely occurs with caries.

Caries of the upper cervical region and the atlanto-occipital joint produces *pains* in the head and neck, vertigo, and even nystagmus (Bergmann), *stiffness* of the neck, and sometimes *crepitation*. The head is held in a fixed position, and all movements are avoided. The earliest symptom of involvement of the roots is the occipital *neuralgia*. Anesthesia of the occipital or upper cervical nerves may occur later. Finally the symptoms of *cervical myelitis* or a *bulbar paralysis* present themselves. Sudden death may occur at any moment.

Diagnosis.—If deformity is absent, the sensitiveness of a certain vertebra, the careful position in which the head or trunk is held, with symptoms of involvement of the cord or nerve-roots, will make a diagnosis. In one of my cases I was able, with Röntgen's rays, to discover the vertebral disintegration and dislocation. If all signs of vertebral disorders fail, youth, tuberculosis of another organ, attacks of fever, together with symptoms of a slow, progressive compression of the spinal cord and roots, show the nature of the disease.

Never inject tuberculin to confirm a diagnosis.

If no deformity exists, the affection may be confused with tubercular myelitis or meningo-myelitis (Raymond).

It often occurs that caries may be confused with tumors or other vertebral diseases. I once diagnosed as caries a sarcoma that had extended from the kidneys to the vertebral column. In cases of this nature, however, the pain is generally more severe and the gibbus develops more slowly.

A simple spondylitis or bone softening may develop from a vertebral trauma, symptoms of which (gibbus, pains, and signs of compression) may appear months or even years afterwards (Kümmel, Henle).

I have seen cases of caries which at the onset were diagnosed *neurasthenia* or *hysteria*. The patients complained of pain in the back; the results of examination for pain on pressure were uncertain and varying; there was slight weakness in the legs, with increased reflexes; in general, the patient appeared hypochondriacal. I also have observed cases of hysterical rigidity of the neck which had been diagnosed caries. Caries of the upper cervical region may be confused with torticollis on superficial examination.

Course.—It is a chronic disease; the symptoms of vertebral disease may exist months or years before spinal symptoms appear. Sometimes, however, the spinal disease is the first signal of any trouble, and the deformity appears later, or the vertebral affection remains latent; or the signs of spondylitis and compression of the cord occur simultaneously. The spinal symptoms progress gradually, to reach their height in about a year, although the paraplegia may have a sudden onset. Spontaneous recovery may result, the abscess discharging externally and the paralysis disappearing; or the paralysis may persist and the caries disappear, or *vice versa*. Sometimes the disease remains stationary; remissions may also occur. In many cases decubitus ensues, and cystitis, polynephritis, septicemia, or general tuberculosis ends the scene.

Prognosis.—More favorable in youthful persons, with partial development or absence of spinal symptoms, when the lesion is a dorsal one, or when good nutrition and strong muscular development exist. Worse when caries commences late in life, attacks a decrepit person, and when signs of a general tuberculosis are present. The prognosis becomes worse the longer the disease lasts.

Billroth cites ninety-seven cases of caries, forty-eight of which died, twenty-two recovered, and eleven were discharged still sick. The records of Reinert's clinic give a mortality of sixty per cent., and thirty per cent. cures. In a case of Loison's, recovery is said to have spontaneously followed an attack of erysipelas.

Gowers observed a case of caries without any spinal trouble, which in later years developed the symptoms of lateral sclerosis. I observed incontinence of the bladder and bowels develop in an eleven-year-old child who had suffered with caries in his fourth year, but who had been apparently well since that time.

Treatment.—1. Preserve the strength of the patient. 2. Avoid all movement of or pressure upon the involved vertebræ.

Order good, fatty, and albuminous food, give cod-liver oil (with phosphorus), and compel the patient to *rest* constantly in bed. He should lie on his back for months, naturally guarding against decubitus by the use of smooth bedclothes, washing of the gluteal region, great cleanliness, and water- or air-pillows. If no results are obtained, extension must be made. This is done most easily in cervical caries. For methods of extension and directions for the use of the various apparatus (Sayres's, Glisson's, Bennet's, Volkmann's, Rauchfuss's, or Hessing's), see books on surgery. Phelps and Lorenz have recommended extension-beds of plaster of Paris; Karewski, a suit of plaster of Paris. The use of setons, cautery, etc., may be in place when other methods fail; but modern views are against a revulsive treatment.

I have had very good results with iodide of iron preparations,—the saccharated iodide of iron and the syrup of iodide of iron. Calcium phosphate is also recommended. Mercury and iodides should, of course, be used in cases that are under the suspicion of being syphilitic.

In 1888 Macewen published his results of *operative treatment of vertebral caries*. His results were accepted with enthusiasm; which has, however, considerably diminished since more extensive experience has shown in how few cases operative interference is really of benefit.

Thorburn's and Chipault's statistics show that out of one hundred and three laminectomy cases, forty-three died soon after the operation, while in only fifteen was a cure effected. The following may be accepted as indications for operation:

1. In caries of the vertebral arches, when conservative treatment does not improve it (Péan's result). 2. When the opening to the congestion abscess leads directly to the seat of the disease.

Schede believes also that, when all other methods fail, laminectomy may be made the *ultimum refugium*.

Calot's forcible *redressement* is one of the newest methods for cure of Pott's deformity, but the German surgeons are still somewhat chary of trying it.

Chipault has also recommended forcible *redressement*, but his method has not been found to be very practical.

These various methods cannot, however, be discussed here. Warm baths, preferably medicinal ones, should be advised. Electrical treatment cannot do much good; it should be tried only in old cases with flaccid paresis or atrophy. Massage and even tenotomy may be beneficial in contractures following an old caries. To guard against remissions, the patient should live hygienically, should avoid falls upon the back, and should protect his back with a small, soft cushion.

CARCINOMA AND OTHER TUMORS OF THE SPINAL CORD.

Carcinoma of the spinal column is generally a secondary metastatic tumor. The primary neoplasm has its seat in the stomach, uterus, mammæ, or other organs. *Sarcoma* and osteosarcoma may originate in the vertebræ, but generally extend to the vertebræ from the surrounding parts. They occur principally in the old, although sarcomata may be found in young persons. These tumors rarely confine themselves to one vertebra, but spread to the neighboring ones, producing a diffuse infiltration or necrosis. They may involve the neighboring ribs or muscles and appear under the skin. A kyphosis is generally produced, though it is not so sharp as that in caries, as more vertebræ take part in the deformity and the tumor itself helps to produce the prominence.

The disease is very painful, the pains being increased by movements, pressure, or concussion of the diseased vertebræ.

When the posterior roots are involved, severe neuralgic pains occur. In carcinoma of the lumbar vertebræ, a severe bilateral sciatica may be the first symptom. Hyperesthesia and local muscle-spasms also are very marked symptoms. In general, the symptoms are similar to those of caries.

Paraplegia may develop very rapidly, the paralysis spreading quickly.

The old age (in carcinoma), the general debility, the detection of a primary tumor, the great pain upon pressure and movement of the vertebræ, the acute-angled or round kyphosis, the spinal-cord and nerve-root symptoms are all factors in support of the diagnosis. Occasionally we are able to palpate the tumor. In doubtful cases we should use X-rays.

In caries the pain is not so great, the patient is generally younger, and there are other signs of tuberculosis. Presence of a primary tumor in other organs always speaks for tumor of the vertebral column. In some cases the vertebral trouble remains latent a long time. I once observed a case in which at first only general diffuse pains were complained of. After a few weeks, symptoms of paralysis in the legs, weakness of the bladder, and disturbed sensibility appeared, the pains growing constantly worse. As syphilis was acknowledged, I made a diagnosis of spinal lues, and ordered the proper treatment for it. Some months afterwards, a deformity appeared in the lower dorsal region, and further developments showed that a sarcomatous growth, probably starting in the retroperitoneal glands, had worn away some of the vertebræ and compromised a large part of the cord. A myelitis or multiple neuritis may develop upon a previous tumor cachexia. Also, in carcinoma, pains may be present in different parts of the body, likewise in the spinal-cord region, which are not due to any tumor in the spinal cord. The diagnosis of metastatic carcinoma of the vertebral column should not, therefore, be made from vague symptoms.

Sarcoma of the vertebral column runs generally a chronic; carcinoma, however, an acute, course.

The prognosis is always a bad one. Operative procedure has not availed much. Trephining of the spinal column, as recommended by Sonnenburg and Horsley, did not do any good in a case of mine in which I tried it. All that can be done is to dispel the pain with morphine and to avoid pressure upon the diseased part.

SYPHILITIC DISEASES OF THE SPINAL COLUMN.

Syphilis rarely attacks the vertebræ. Occasionally we find the formation of *exostoses*, which may extend and involve the nerve-roots and the spinal cord. Gummata may also occur. A syphilitic form of spondylarthrosis has been occasionally observed.

In one of my cases large, irregular exostoses were palpable in the upper cervical region; there were paralysis and disturbed sensibility in all four extremities. As syphilis had existed, I put him on an energetic inunction cure, which in time brought complete recovery.

ARTHRITIS DEFORMANS OF THE SPINAL COLUMN.

This occurs rarely. It may involve the whole spinal column, may produce calcification of the intervertebral cartilages, new bony formation on the spinal processes, and thus cause complete ankylosis. It also may confine itself to a definite region,—for example, the cervical part, when the head and neck will be fixed and the chin inclines towards the breast.

We find *spinal pains*, increased on movement, and also, when the nerve-roots are involved, *radiating pains* (intercostal, brachial, and crural neuralgia) and *partial paralysis with atrophy* in the region of the muscles of the extremities. The spinal cord is not often involved.

It runs a chronic course with remissions and exacerbations.

We make our diagnosis upon the presence of arthritis elsewhere, the immoblization of large sections of the spinal column,—an ankylosis which does not disappear under chloroform,—and upon the symptoms on the part of the nerve-roots. Palpation often leads to a certain diagnosis. Bechterew describes a growing together of the entire spinal column, the anatomic nature of which is not certain. Strümpell has described similar cases. They were probably all cases of arthritis deformans.

Massage, local applications of iodine, and medicinal baths (sulphur or alkaline) may be beneficial.

A sulphur bath may be made by adding to the water fifty to one hundred and fifty grams of sodium sulphate, fifty to one hundred grams of potassium sulphate, and twenty to thirty grams of crude sulphuric acid. Potassium iodide may be given internally. I only observed one case in which an acute articular rheumatism had involved the spinal column and produced severe symptoms of the involvement of the nerve-roots (paralysis with atrophy). The sulphur baths of Aachen, Baden, Nenndorf, Lenk, Kainzenbad, etc., are of benefit.

B. DISEASES OF THE SPINAL CORD OF MENINGEAL ORIGIN.

SPINAL MENINGITIS.[1]

Acute spinal leptomeningitis is rarely a primary, isolated disease of the meninges of the spinal cord. When it is not of traumatic origin, it is generally the epidemic, cerebro-spinal form, or a purulent meningitis resulting from septicemia which occurs in the puerperium, or following purulent wounds or the acute infectious diseases.

The spinal meninges also take part in tubercular inflammations of the cerebral membranes; a traumatic purulent meningitis of the cerebral meninges may also extend to the spinal cord. Otitic meningitis has a special tendency to extend to the spinal meninges.

Pathological Anatomy.—A serous, fibrinous, and purulent exudate follows the hyperemia, and is deposited in the meshes of the pia and arachnoid, clouding the cerebro-spinal fluid. The membranes are covered with a semi-solid or purulent exudate, at some places bound together by it. In the *tubercular* form pus is absent. The exudate is slight and gelatinous, and we find eruptions of miliary tubercles upon the arachnoid and the inner surface of the dura mater.

---

[1] The inflammations of the external surface of the dura mater are of no clinical interest, nor are perimeningeal abscesses, and these therefore will not be discussed.

These alterations generally spread over the whole meningeal surface. In the cerebral forms the process, as a rule, does not extend lower than the last cervical vertebra. In general, the membranes on the posterior surface are more involved than those of the anterior. The nerve-roots are also covered with an exudate, and the spinal cord is often involved in its peripheral parts (*peripheral myelitis*).

Symptomatology.—In most cases the cranial symptoms are so prominent that the spinal symptoms are hidden. The rarely observed isolated spinal meningitis has the following symptoms: onset with chill and a high fever of irregular type; marked *pains* in the *spinal column*, increased by movements; *radiating* pains in the extremities; *stiffness of the back; opisthotonos; tonic spams* of the muscles of the *abdomen, breast*, and *extremities*, with clonic spasms on attempts at movement, etc.; hyperesthesia of the skin of the trunk and limbs and increased reflexes. Percussion of the lumbar muscles produces a brisk retraction of the lumbar vertebræ. Kernig's symptom consists in inability to extend the leg in a sitting position on account of spasm of the flexors. Incontinence of the urine often occurs. If death does not result, *paralysis* follows; paraparesis, even paraplegia, lessened sensibility, weakness of the bladder, and marked deep reflexes, disturbed respiration, an irregular heart, and oculopupillary symptoms may all occur.

It runs an acute course, may be foudroyant, and has a bad prognosis. Death may result in a few days or weeks. Recovery occurs sometimes in the epidemic and rarely in the otitic type. Partial recovery may occur, the paraparesis, bladder weakness, etc., remaining.

Concerning the **treatment** and further details than are given here, see the chapter on Cerebral Meningitis.

In the first stage absolute rest in a proper position is of great importance. The patient should be placed upon the side or face. This position is not only comfortable, but permits most easily the local treatment. A diaphoretic and purgative treatment should be commenced from the onset. Use wet packs; apply dry cups or leeches to the back. An ice-bag to the spinal column can be used if it does not produce an uncomfortable position. Inunctions of mercurial ointment as well as the giving of calomel internally are recommended. In the latter stages apply cantharides to and use a cautery on the back, and order hot douches. There is still some doubt as to the benefit of lumbar puncture in such cases. (See chapter on Cerebral Meningitis.) Phelps, among others, has reported a case cured by its use.

CHRONIC SPINAL MENINGITIS.

Simple chronic spinal meningitis is a disease which has little clinical importance, as far as our experience goes. At least, exact clinical types, confirmed by autopsy, are not found in the literature. It forms, however, a common and unexpected post-mortem discovery in persons who have died from other diseases. It may thus be accidentally discovered accompanying tabes, myelitis, etc. Alcoholism, senility, spinal concussion, continued influence of cold, severe bodily exertion, are all given as etiological factors. Until recently chronic spinal meningitis and meningomyelitis were looked upon as the anatomical basis of "railway spine." This view was, however, a false one.

Acute meningitis may become chronic. The membranes become clouded, thickened, and have grown together and to the spinal cord.

In the symptomatology built up *a priori*, the most prominent symptoms are pain in the spinal region, slight paralysis, combined at times with atrophy and disorders of coördination.

It is certain that the old neurologists confused this disease with neuritic, myelitic, and hysteric affections.

A circumscribed form of tubercular meningitis has occasionally been observed.

There are two types of chronic meningitis which deserve some notice:

1. *Pachymeningitis cervicalis hypertrophica.*
2. *Pachymeningitis and leptomeningitis chronica syphilitica.*

PACHYMENINGITIS CERVICALIS HYPERTROPHICA (CHARCOT AND JOFFROY).

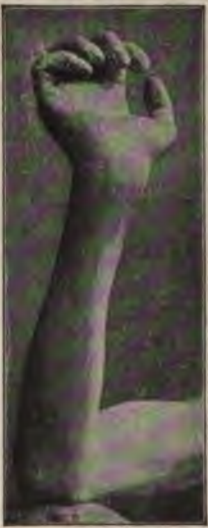
This is a chronic inflammation, especially of the inner layer of the dura mater, which produces a layer-deposit of fibrinous tissue, and consequently a thickening of the dura (to five or ten times its normal thickness). The newly-formed, strata-like, hard, often ossified, membranes bind together the meninges, nerve-roots, and spinal cord, and the latter may be so involved that atrophy and sclerosis result. The soft membranes of the cord are often the starting-place of the inflammation. At first only the peripheral layers of the cord are affected, but soon the whole cross-section becomes more or less involved, partly by reason of the compression, partly from the extension of the inflammation through the vessels and the pial septa to the inner parts of the cord. Lymphatic congestion may also be a factor, and occasionally cavities are formed in the cord.

This process confines itself, as a rule, to the dura mater around the lower cervical region, but may extend much farther, and even involve the medulla and pontine region (Adamkiewicz, Wieting).

Etiology.—Cold, over-exertion, injuries, and *syphilis*, all have been given as causes. Syringomyelia has also been asserted to be superinducive in some cases, while many deny that the disease is an independent one. Syphilis is certainly at play in some, if not in all, cases.

The first **symptoms** are produced by the meningitis itself, as well as by the compression of the posterior roots. Pains in the neck, between the shoulders, and in the back of the head, a feeling of tension and perhaps actual rigidity, sensitiveness of the cervical vertebræ to percussion, paresthesia and neuralgic pains, especially along the *ulnar* and *median* nerves, tremor, and slight muscular stiffness,—all these are symptoms of the neuralgic stage, which may continue weeks and months. It is followed by *paralysis*, which is of *neuritic origin*. We find then hypesthesia and even anesthesia in the zones of the skin innervated by the affected nerves, and degenerative paralysis in the muscles supplied by them, especially in the small muscles of the hand and the flexors of the hand and fingers. The lack of resistance to the action of the extensors produces a peculiar and almost pathognomonic position of the hand,—over-extension of the wrist-joint, extension of the basal and flexion of the middle and end phalanges (preacher's hand). (Fig. 125.)

FIG. 125.



Position of the hand in paralysis of the muscles supplied by the median and ulnar nerves. Type of "preacher's hand," incompletely developed.

Oculopupillary symptoms may occur (first dilatation, later contraction), but there is nothing characteristic about them.

In a third stage, which is not sharply defined from the second, we find symptoms of a break in continuity in the lower cervical cord, such as spastic paralysis of the legs, anesthesia, bladder trouble, etc. The pains decrease as the disease advances. The disease may cease to progress at any stage, and recovery may even take place (Charcot, Berger, Remak). In general, however, a very *grave prognosis* should be made.

When the dorsal cord is involved, intercostal pains usher in the disease; then comes anesthesia in the same area, and finally paraplegia. If the process affects the pons and medulla oblongata, symptoms showing this involvement occur,—*e.g.*, in a case of mine I found bilateral impairment of the hearing and *tachycardia;* in a case of Wieting, bulbar symptoms were present.

According, then, to the localization of the lesion, the symptoms may be variously modified, making the diagnosis often uncertain; which is not the case, however, when the focus is at the common seat of the lesion.

Treatment.—*Revulsives*, as applications of *iodine* and the use of *a cautery* in the upper cervical region, are in place. Where there is a question of syphilis, mercury and the iodides must be used, and are justifiable in other cases also. Warm baths and sweat-cures often bring relief. In one of Remak's cases, seen by me, galvanic electricity produced good results.

CHRONIC SYPHILITIC MENINGITIS (ARACHNITIS GUMMOSA, ETC.) AND THE OTHER DISEASES OF THE SPINAL CORD OF TRUE SYPHILITIC ORIGIN.

Syphilis of the spinal cord generally originates from the meninges. Syphilitic meningitis and spinal lues have about the same symptoms, and may be discussed together.

Syphilis plays a prominent part in the etiology of diseases of the spinal cord. It is not our purpose here to discuss those spinal diseases which have only an etiological connection with lues, but rather those which we regard in a pathologico-anatomical sense as specific spinal-cord diseases.

The prototype of all is the universal *syphilitic meningitis*. It commences in the soft membranes of the spinal cord, more rarely from the inner surface of the dura.

Here granulation tissue forms, which extends over the surface and leads to a clouding and thickening of the membranes, binding them together and to the spinal roots and cord.

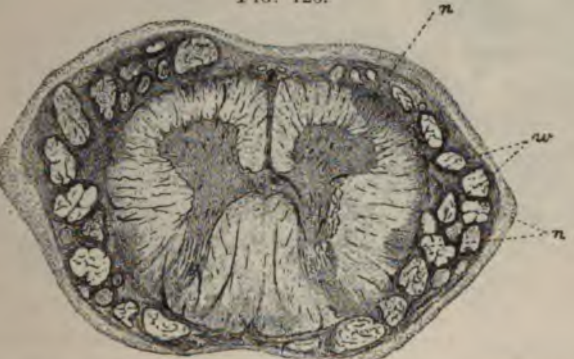
Macroscopically we notice that the meninges are thickened and covered with a spotted, gelatinous, or fibrous tissue. As long as the dura is unopened, the cord appears in many cases to be greatly swollen *in toto* or over a large area. If then an attempt is made to separate the dura, the existing *adhesions* hinder greatly. After doing this, the gray-yellow, soft (or, in places, hard) deposits appear, arranged irregularly, either diffusely or (more rarely) similar to a circumscribed tumor. Cross-sections reveal anomalies, which must, however, be more closely studied *microscopically*.

We find in such an examination a meningeal affection of varying intensity at different heights. (Figs. 126 to 128.) At one place the meninges may be greatly increased in size by a thick cellular deposit, which is also highly vascular. At another place the meningitis is merely indicated, and a hard, fibrous tissue is found which caused the proliferation. The process does not extend equally over the circumference of a cross-section; it is especially pronounced, in many cases, in the region of the posterior cords. In the newly-formed tissues local gummata have developed. (Fig. 128.)

The spinal roots may be normal or infiltrated or atrophied (Figs. 126 and 127).

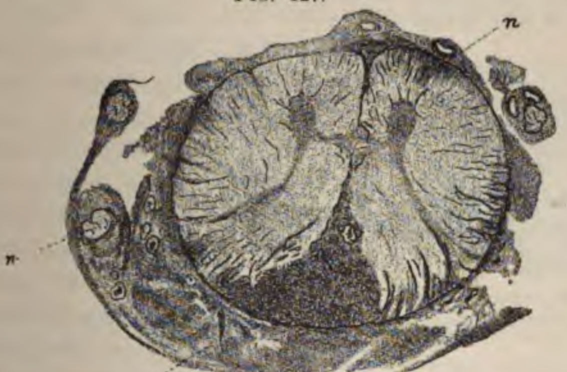
The spinal cord itself shows a slight *inflammation* of its *periphery*, but in some places it may be heavily involved, the granulation tissue sending cord-like processes into the spinal cord from the periphery, which, as they extend inward, produce inflammation and atrophy of the surrounding parts. At other places the cord is simply *softened*. The blood-vessels are generally affected, and in some cases may be the starting-point for the disease (Raymond, Lamy, Schmaus, Rosin).

FIG. 126.



Syphilitic meningo-myelitis. *n*, neoplasm; *w*, roots. (Carmine stained.)

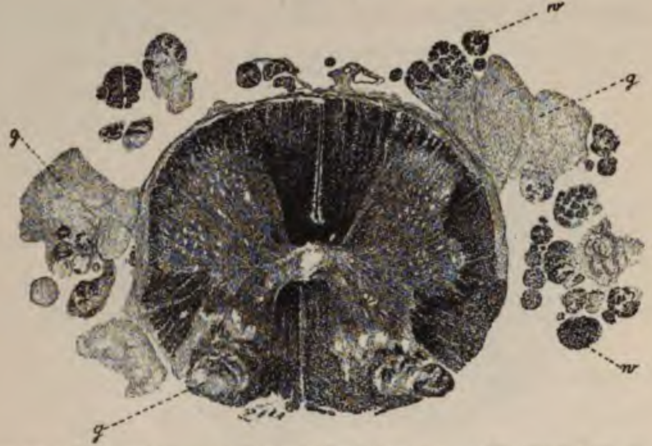
FIG. 127.



Syphilitic meningo-myelitis. *n*, neoplasm which surrounds the extra-medullary roots and enters the medulla at different places. (Carmine and alum-hematoxylin.)

The arterial walls are thickened to *obliteration;* the smaller ones may be completely consolidated, and in the veins also we may find a *phlebitis obliterans* (Greiff). Considerable hemorrhage and formation of cavities have been observed occasionally.

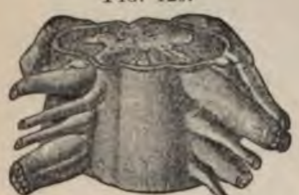
FIG. 128.



Gummatous meningitis and gummatous neuritis of the roots. *g*, gummatous tumor. (Weigert's stain.) (After a Siemerling's specimen in my collection.)

A simple *disseminated* and *diffuse myelitis*, a *poliomyelitis* (?), may also develop from syphilis. In a few cases an *isolated* gummatous tumor was found in the spinal cord (McDovel, Wagner, Wilks, Osler, etc.). Kahler (Fig. 129) has described a gummatous neuritis of the roots of the spinal and cranial nerves.

FIG. 129.



Syphilitic neuritis of the roots of the spinal cord. (After Buttersack.)

Symptomatology.—The symptoms of spinal syphilis follow rather soon after the infection. In many cases the disease appears before the end of the first year,—in one of Nonne's, after only three months, in most cases, however, within six years. *Hereditary syphilis* may also produce spinal disease soon after birth or later in life. The less complete the treatment of the syphilis, the earlier the spinal lesions seem to appear. Traumata, colds, and infectious diseases help the process. A clinical picture that will suit all cases of spinal syphilis cannot be made. It varies according to the extent, intensity, development, diffusiveness, and location of the lesions.

Certain peculiarities of its course and certain phenomena are, however, *characteristic* enough to aid materially in the making of a diagnosis. The meningeal trouble produces *pain* in the back, at times marked and persistent, on other occasions slight, and is not always accompanied by hyperesthesia.[1] Charcot believed the pain to be more severe at night.

The compression and infiltration of the posterior roots causes *radiating pains* in certain nerve-tracts: *girdle pains,* and neuralgic pains in the extremities. These may be very persistent and diffuse, and may be absent in other cases. If the anterior roots of the cervical and lumbar prominences are involved, *paralysis with atrophy* of the arms or legs occurs. It is generally a *partial* one, and is confined to a few muscles or muscular groups.

Dejerine and Thomas, for instance, have described a syphilitic spinal meningitis at the origin of the eighth cervical and first dorsal roots with the symptoms of Klumpke's paralysis (which see). I observed in some cases a tremor with rapid oscillations, which was perhaps induced by irritation of the anterior roots.

The most important symptoms are produced by involvement of the spinal cord, and are generally indicative of a partial *break in continuity,* consisting in paralysis of a leg, in *spinal hemiplegia,* in *Brown-Séquard's paralysis* (rarely complete), or in *paraparesis,* one leg being weaker than the other. It may be a spastic or atrophic and flaccid paralysis, though generally the former.

The *bladder* and *rectal functions* are usually impaired, as is also the sensibility. All qualities of sensation may be disturbed singly or in association.

This *association* of *meningeal, spinal,* and *spinal-root symptoms,* the *multiplicity* and *incompleteness* of the symptoms, their *instability,* coming and going, with exacerbations and remissions,—all these are indicative and characteristic of the syphilitic nature of the process. I have seen the knee reflexes absent, in a few days normal, and after some time increased. This fluctuation of symptoms is abrupt, often changing from day to day the whole clinical picture. *Ataxia,* if present, may vary in the same way.

Often a cerebrospinal meningitis is found *present at the same time,* the cerebral symptoms overshadowing the spinal. Gerhardt calls attention to a triplegia which occasionally occurs,—*i.e.,* a paralysis of three extremities, a combination of hemiplegia with paraplegia.

Some cases resemble acute, subacute, or chronic *myelitis,* and may be progressive in character.

---

[1] Simple meningeal irritation is said to be a transient symptom of the second stage of syphilis.

Erb has described as syphilitic *spinal paralysis* a group of cases which develop gradually a spastic paresis of the legs, with increased reflexes and disorder of the bladder and sensibility. Although the gait is spastic, the muscular contractions are slight. Its course is chronic, with a tendency to improvement, remissions, and complete cessation of all symptoms.

These cases may be mostly syphilitic meningo-myelitis with particular involvement of the dorsal cord, and often only in a certain stage of the disease.

Nonne believed that chronic syphilitic meningitis was a system disease of the cord produced by the syphilitic toxine. (Trachtenberg claims the same.)

In some cases, as I have shown, the similarity with tabes is so great that I have called them *syphilitic pseudo-tabes*. In these cases the specific process extends from the meninges to the posterior cord and roots, and produces Westphal's symptom, ataxia, lancinating pains, bladder-weakness, etc. Paralysis of the ocular muscles, pupillary rigidity, laryngeal paralysis, nervous deafness, etc., were found, as also anesthesia of the trigeminal region, produced partly by atrophy and partly by a basal meningitic and gummatous-neuritic involvement of the cranial nerves and their roots. The symptom-complex of spinal meningitis may also remind one of that of gliosis, of amyotrophic lateral sclerosis, etc. Bechterew described a syphilitic cerebrospinal focal sclerosis, but acknowledged that it was not identical with multiple sclerosis.

The region of the *caùda equina* may also be involved. The symptoms are radiating pains in the region of the sacral nerves, especially the pudendal, also of the bladder, rectum, perineum, penis, etc. We also have anesthesia in the same region, paralysis of the bladder and rectum, and impotence. I have seen such a case cured by the inunction treatment.

Differential Diagnosis.—Without a history of syphilitic infection, one examination will not suffice for the making of the diagnosis. Only after repeated observation is this possible. On the other hand, the possibility of every diffuse (one could almost say every) disease of the spinal cord being of syphilitic origin must be remembered. Especially characteristic are its step-like character, the incompleteness of the symptoms, the oscillation of individual phenomena, the intercurrent commencement of cerebral symptoms, and the diffuse and disseminated character of the disease. These characteristics do not occur in simple myelitis or in compression myelitis. Nor should they be expected in every case of spinal syphilis. Multiple sclerosis and combined disease of the lateral and posterior tracts may be confused with spinal lues, but the characteristic symptoms of these diseases, as given under their respective headings, will serve to distinguish them.

Course and Prognosis.—A few cases are acute, and death or recovery results in a few weeks or months. Most of the cases run a chronic, remittent course. They take months, or even years, to develop, then recover or remain at a stand-still until, without any external cause or from some injurious movement, a relapse occurs.

The prognosis is better than in the diffuse diseases of the spinal cord of non-syphilitic origin. Complete recovery, however, takes place in only a small percentage of cases. Clinically, the prognosis is the better the less the symptoms of paralysis are developed and the shorter time that they have existed; anatomically, the less the spinal cord itself is involved. Recovery is not excluded in those cases in which a more or less complete break in continuity is present, in which there exists a paraplegia. The symptoms, however, must not have existed longer than a few months. Whenever a secondary degeneration or atrophy has taken place, recovery is out of the question; but before that—*i.e.*, before the disease has existed many months—this is possible. In those cases which have a progressive course and soon end in death, a diffuse myelitis or diffuse softening is found; here also belong severe cases of disseminated cerebrospinal sclerosis, in which the medulla oblongata (vagus) or the cerebrum is involved. In most cases only an *improvement* can be expected: the meningeal and root symptoms disappear under treatment, but certain signs of spinal disease, above all others the spastic paresis, bladder weakness, etc., persist. Relapses are always to be looked for.

Treatment.—In every case an antisyphilitic treatment must be given without delay. Give inunctions of three to five grams of gray mercurial ointment, and prescribe iodide of potassium in gradually-increasing dosage, or in large doses from the beginning (two hundred to three hundred grains). It is generally not possible to produce a complete cure; the improvement advances to a certain point, and then a stand-still occurs. We may even use inunctions of two hundred and fifty to three hundred grains from the first. The treatment then must be stopped for a time, to be resumed after some months, in relapses, or upon the appearance of new symptoms. Even if recovery has taken place, a repetition of the treatment once a year is advisable. Guard against cold, traumata, over-exertion, and *sexual excesses*,—as spinal douches, mountain waters, hot baths, sexual excess, etc., have been known to cause relapses. The diet should be attended to. Marriage should be forbidden, even after recovery. Sometimes the mercurial treatment only produces its results by the use of a hydropathic cure at the same time or afterwards. The baths of Aachen, Nenndorf, and Weilbach (Hot Springs, Mount Clemens, etc.) are especially popular. A sojourn in the South has several times produced the desired results.

In the spastic forms, after the acute stage has passed, the patient may be permitted to do a little walking, though he should not be permitted to tire himself. A cauterization of the spinal column, electricity, mild massage, passive movements in a warm bath, are all good methods of treatment after the disease has run its course, and after the specific treatment becomes inutile.

C. PRIMARY DIFFUSE DISEASES OF THE SPINAL CORD.

MYELITIS.

Our increased knowledge of the pathology of the spinal cord has restricted the use of the term myelitis, so that we now understand by it only the *diffuse and disseminated inflammations* and *softening processes in the spinal cord*, and not, as the name implies, all forms of spinal-cord inflammation. Still, however, our conception of the term is not a precise one.

Myelitis occurs at all ages, but especially in persons of middle age. A true myelitis occurs much more rarely in childhood than does a poliomyelitis. Senile spinal cord paralyses demand separate treatment.

Etiology.—Over-exertion, psychical emotion, sexual excesses, traumata, and especially exposure to cold, were formerly given as the causes of myelitis. They are now regarded simply as predisposing factors. *Infection* and *intoxication* are now known to be the important factors. It is certain that the *acute infectious diseases* may produce it. Observations of Gubler, Imbert, Westphal, Ebstein, and, later, Leyden-Renvers, Lenhartz, Putnam, and others, have confirmed this. Bruns traced it once to varicella, Specker to a septic infection, Kowalewski to inoculations against hydrophobia. It may probably occur as a primary infectious disease, as the observations of Küssner and Brosin, Achard and Guinon, and others, seem to show.

The forms of myelitis occurring in the puerperium and in *pregnancy* seem also to be of an infectious nature.

Gonorrhœa may produce a myelitis (Gull, Barrié, Leyden, Dufour). I observed it soon after vaccination in one case. It may also follow tuberculosis and syphilis. We do not refer to the true syphilitic or tubercular diseases of the spinal cord previously described. It is simply a myelitis which can neither clinically nor anatomically be recognized as specific.

Malaria may also cause it. The connection of myelitis with diseases of the urinary apparatus (paraplegia urinariæ) and with an ascending neuritis is not known, though it is certain that it follows them. I have never succeeded in convincing myself that a neuritis may ascend a nerve-trunk to the spinal cord and produce inflammation there. Others—Charcot, Leyden, Bompard, and Shimamura—have described cases which they trace to such an origin.

The micro-organisms of the infectious diseases themselves are rarely found in the spinal cord, more often only streptococci and staphylococci, so that we are inclined to think that the myelitis results from a mixed or secondary infection (Grasset). Whether the myelitis is caused by the bacteria or their toxines is also unknown.

Simple *intoxications* with CO_2, CS_2, chloroform, nitro-benzine, and illuminating gas have been observed to produce diseases which probably belong here.

Symptoms.—The typical form of myelitis is a *transverse myelitis.* We will suppose that the case is fully developed and that its seat is in the dorsal region (*myelitis dorsalis*). This is the part of the cord which is most often involved. We find then the symptoms which would be produced by division of the cord at that height; namely—

1. *Paraplegia.*—The legs are paralyzed. It is generally a *spastic* condition with *increased deep reflexes.*

They are at first extended, but later *flexion contractures* may be present, in which the legs are bent at the knee-joints and drawn towards the abdomen. More often, however, spontaneous movements and *twitchings* occur which bring the legs at times to an extended, at other times to a flexed position. The contracture may be so marked as to prevent the knee reflexes from acting.

2. *Anesthesia.*—The sensibility is lost in the legs and on the body, according to the seat of the disease, and, as a rule, for all qualities. The upper limit of the anesthetic region is generally bordered by a zone in which the patient at times experiences *girdle pains,* at which place a slight hyperesthesia also may be objectively detected.

3. *Reflex excitability* is generally increased in the legs. In those cases in which the lesion involves the whole cord Bastian claims that other laws govern the reflexes. (*Vide* chapters on Spinal Localization and Reflex Action.)

4. *Paralysis of the Bladder and Rectum.*—The patient is no longer able to influence the function of these organs, nor has he any feeling in them. This results in retention or incontinence of urine and feces. Impotence also exists. Priapism occasionally occurs or erections occur during catheterization.

5. *Decubitus.*—Pressure, loss of sensation, and uncleanliness,—these factors combine to produce an ulcer, generally in the region of the os

sacrum, or of the trochanters, occasionally in the toes, etc. It may be superficial or so penetrating as to lay bare the bone. Trophic disturbances are probably also at work here. Pemphigus and other skin-diseases are often present.

Vasomotor and secretory disorders may also occur,—edema, arthropathies, anidrosis, etc.

The muscles retain their volume and electrical excitability, even after the paralysis has existed for some time.

MYELITIS LUMBALIS OR LUMBOSACRALIS.—In this region the paralysis is a *flaccid* one. The *deep reflexes* are lost; the *skin reflexes are abolished;* the anesthesia does not extend far up the trunk; there is no girdle sensation, but radiating pains in the paths of the nerves of the extremities. The paralysis of the bladder and rectum is very pronounced.

If the upper lumbar cord is not involved, the knee reflex is normal or increased, because the ileo-inguinal, crural, and obturator nerves are not involved. A primary myelitis limited to the conus terminale produces paralysis of the bladder and rectum, impotence, anesthesia of the anal, scrotal, and perineal regions, and of the penis and the upper inner surface of the thigh, and perhaps a degenerative paralysis of the sciatic region.

CERVICAL MYELITIS.—If the *lower cervical* region is involved, we observe: (1) *atrophic paralysis of the arms;* (2) *spastic paralysis of the legs;* (3) *anesthesia of both arms and legs, as well as of the trunk;* (4) *oculopupillary symptoms.* The involvement of the abdominal and intercostal muscles, which may occur, causes difficult breathing. In myelitis of the *upper cervical* region the atrophy of the arms is absent; they are in a spastic condition. Another symptom noticed is paralysis of the diaphragm, and if the process ascends still higher, the medulla oblongata becomes involved.

INCOMPLETE TRANSVERSE MYELITIS.—When the transverse lesion is an incomplete one, we have only slight symptoms (paresis in place of paralysis, hypesthesia for anesthesia), or some of the above symptoms may be altogether absent. Partial sensory paralysis occurs sometimes, and still more rarely Brown-Séquard's symptom-complex. It may be regarded as a rule that all signs of a transverse break in conduction are present if only a part of them are indicated.

The myelitic process may involve a considerable area,—the whole dorsal or a part of the dorsal and lumbar cord. The resulting modifications in the symptomatology do not deserve a special description.

MYELITIS DISSEMINATA.—In this form small foci are scattered over the whole cord, and may involve the medulla, pons, or brain. This is pre-eminently the type which follows the infectious diseases. It is also often of toxic origin. The symptomatology varies according to the seat and extent of the lesions; symptoms of involvement of the brain and medulla preceding or accompanying the cord symptoms. Many cases are atypical; some resemble acute multiple sclerosis. In a few of these the ataxia and intention tremor were so prominent that they were called cases of *acute ataxia*. It should not be forgotten, however, that the so-called acute ataxia may also be due to a peripheral neuritis. I have several times seen this type of myelitis change into multiple sclerosis. Cerebral symptoms—especially in the beginning—may be very prominent. Choreic phenomena have also been described.

We have only discussed the advanced stage of myelitis; the **development and course** have not been spoken of.

As a rule myelitis comes on abruptly and develops rapidly. In some cases the development was apoplectiform. The temperature may be considerably increased in these acute cases. The patient has a chill, has a feeling of formication in the legs, anesthesia develops, he feels his legs getting weaker, which turns to paralysis, etc. He has little pain. The disease often comes on *subacutely*, or it may be *chronic*. Cases which are thought to be chronic myelitis generally turn out to be multiple sclerosis with predominating spinal symptoms. A chronic myelitis does, however, occasionally occur. In some cases the myelitis takes an insidious course. Weakness in one leg is felt or in both from the beginning, which is almost always combined with stiffness. It increases gradually. Sensory disturbances come on or were present from the first, paresthesia, then hypesthesia also, which may increase to anesthesia. Other myelitic symptoms develop in a similar manner.

The prognosis is always a doubtful one. Recovery, improvement, remissions and relapses, continued progress of the disease till death results, may all take place.

If it follows an acute infectious disease, the chances for recovery are favorable, the type following gonorrhœa always getting better under proper treatment.

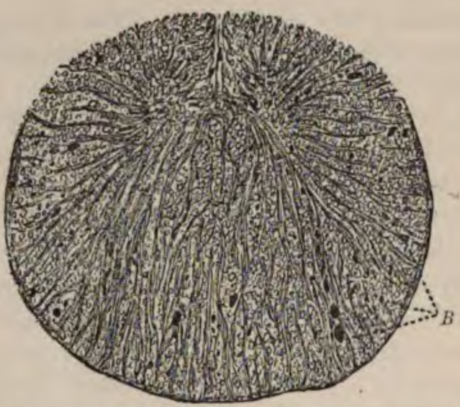
The disseminated type succeeding variola, typhoid, erysipelas, influenza, etc., and which resembles acute ataxia, frequently undergoes recovery. Forms of spinal paralysis of an *intermittent* character and malarial in origin, generally recover. The prognosis of syphilitic myelitis is not exactly unfavorable. A myelitis resulting from sepsis or tuberculosis or in the puerperium has a bad prognosis. The acute incomplete types of myelitis show a more favorable prognosis than the insidious progressive cases or the cases of complete transverse myelitis.

I have seen cases complicated by meningeal and neuritic symptoms which recovered.

Acute decubitus is always a grave omen, as is also complete paralysis of the bladder and rectum. Slender, weak individuals and the aged bear the disease better than the robust. The longer the symptoms have existed, the worse is the prognosis. Death generally results from decubitus or cystitis.

Pathological Anatomy.—The cord feels soft to the touch at the place where it is diseased, and on cross-section it is noticed that in the affected parts the gray and the white matter are hardly distinguishable from each other and are of a red-yellow (first stage) or grayish-yellow color. If a small, fresh piece is placed under a cover-glass and examined microscopically, innumerable granular cells will be found. One or more myelitic foci are seen scattered throughout the cord, and a secondary degeneration is often noticed macroscopically. After hardening in potassium bichromate this condition is still more evident, the diseased parts showing an intense yellow color.

FIG. 130.



Cross-section of the spinal cord in myelitis. Terminal stage. *B*, blood-vessels. (Carmine stain.)

After section and mounting, we find, *microscopically*, a *proliferation* of the *blood-vessels*, *swelling of the axis-cylinders* and the cellular elements, a *puffing* of the cord, later a *degeneration* of the cord, a heaping-up of *granular cells* and *fat-drops*, and finally a *proliferation* of the *neuroglia*. (Fig. 130.) Cysts may occasionally form.

Treatment.—The treatment is not a thankful one. *Absolute rest* is necessary in the first stage; even the excretions should be passed in bed,—with careful regard to cleanliness, of course. A *diaphoretic treatment* is advisable, if it is a post-infectious disease. Local applications of iodine, or counter-irritation with a *vesicant*, may be ordered, being careful not to put it upon an anesthetic zone. Salicylates are of benefit. Use mercury and potassium iodide in specific cases. The view, still expressed here and there, that non-specific cases are also benefited by mercury is not supported by experience. If malaria preceded the attack, give *quinine* and arsenic. I have never had any results with ergot. Strychnine injections may be used for the paralysis. Hot baths should be avoided. Wet packs are of benefit after the first stage is passed.

Warm baths may be used after the disease has ceased progressing. In chronic cases, which have run their course, a stay in one of our numerous bath-resorts is of benefit. Teplitz, Wildbad, Gastein, Rehme, Nauheim, etc., in Germany, can be recommended in this disease; high temperatures must be avoided. Sea-bathing is *contraindicated*. To prevent decubitus, water-pillows should be used, and attention must be paid to the position in bed, changing it repeatedly and seeing that there are no wrinkles in the bedclothes, etc. The catheter must be introduced with care. Use a urinal or cotton to prevent the scrotal region from being irritated by the urine. The bowels should be moved every other day. There is no remedy against the *spontaneous contractions*, even morphine often failing. Hyoscin may do good.

A *permanent bath* in such cases has done good service. Electricity should be used only in the last stage, or in convalescence, when passive movement and massage likewise procure good results.

SENILE PARAPLEGIA.

In old age a motor weakness of the lower limbs occurs which Démange refers to a process allied or similar to an arterio-sclerosis of the spinal vessels, with sclerotic alterations in their vicinity. As a rule, we find spastic paraparesis, which may increase to contracture. The arms can also be involved, or the sensibility and sphincter functions may be impaired. The brain may be affected by the arterio-sclerosis, causing dementia, dysarthria, etc. Spastic paraparesis of the aged may also be produced by small cerebral foci which involve both sides.

I saw several cases in which there were present symptoms of paralysis agitans, in addition to the spastic paraparesis. The tremor was not typical, however.

MULTIPLE SCLEROSIS, DISSEMINATED SCLEROSIS, CEREBROSPINAL MULTIPLE SCLEROSIS.

This is a disease of *youth*. It may occur later in life, but rarely later than the thirtieth to the forty-fifth year. Nor does it often manifest itself before the fourteenth year. Eichhorst, not long ago, claimed to have found anatomical evidence of multiple sclerosis in an eight-months' child whose mother was affected with that disease.

The *acute infectious diseases* may produce the disease (Marie), especially typhoid, variola, scarlatina, and morbilli. Influenza may also cause it, or cholera, diphtheria, whooping-cough, and acute articular rheumatism. I saw it once follow the puerperium. I have also called attention to *intoxications* from metals as a causative factor. Whether the cases observed by Schlockow in zinc-workers are to be placed here is doubtful. I observed it follow carbonic dioxid poisoning in one case. Gerhardt traces a case to poisoning from mercury. Syphilis, however, has nothing to do with multiple sclerosis, though a form of cerebrospinal lues with disseminated foci has been described. Some cases seem to be of traumatic origin, others are apparently due to sudden excitement. In many cases no cause can be discovered.

Symptoms.—*Typical* (Charcot's) *clinical picture.* The description refers to the developed stage.

The patient complains of weakness in the legs, with stiffness, of tremor, of vertigo, and, more rarely, of disorders of vision, headache, pains in the legs, disturbance of speech, etc.

The symptoms observed *objectively* are: motor weakness of the legs, with *muscular rigidity*, the deep reflexes being increased, passive movements hindered, etc.

The gait is a *spastic paretic* one; it may, however, be modified by incoördination, tremor, ataxia, etc. When the weakness becomes pronounced, walking may be impossible. Generally, and often early, it is the uncertainty—the cerebellar incoördination—which influences the gait, so that there is, in addition to the spastic paresis, *reeling*, with danger of toppling over as soon as the patient attempts to walk quickly or to turn.

Active movements are accompanied by a *tremor*, which is absent when the body or limbs are at rest; it accompanies all *voluntary* movements; at times also the reflex and automatic ones. Its oscillations are irregular, and of such wide amplitude as to justify the name *wabbling*. It occurs especially in the large muscles of the trunk and extremities. The whole extremity trembles, making the amplitude of each swing relatively greater. They are relatively slow,—about five to six in a second. The phenomenon is badly called an *intention-tremor*. The tremor is not always accompanied by motor weakness; the arms may retain their strength notwithstanding the pronounced tremor. In later stages, however, we generally find paresis and rigidity, generally more marked in the legs than in the arms.

Another noteworthy symptom is the *nystagmus*. When the patient looks directly ahead, but especially on looking sideways, rhythmic twitchings of the eyeball are observed, which always conduct it from its extreme position to one of rest. Only very noticeable nystagmus is of any diagnostic importance, slight grades being often present normally. Sometimes the twitchings turn the eyeball like a wheel. Kunn has described a tremor which occurs when fixing the eye upon anything.

Disorders of Vision.—The optic nerve is involved in a large percentage of cases. A *partial optic atrophy* is the condition generally found; a pallor of one side, generally the temporal half of the fundus (Uhthoff). *Complete atrophy of the optic nerves rarely occurs. Neuritic* changes may precede the atrophy.

Resulting from this atrophy we have decreased vision (which rarely progresses to blindness), an irregular decrease of the visual field, or a central scotoma for white and colors or only achromatopsia. The similarity of the ocular disturbance with that of intoxication amblyopia probably denotes that poisons are etiologic factors in this disease. The visual disturbance may remit, decrease, or remain stabile. Vision may also be impaired without there being any alterations noticed ophthalmoscopically.

Scanning Speech.—Speech, in developed cases, is slow, the words are jerky, the syllables being separated by pauses. The patient speaks something like a child spelling his alphabet. This *syllabic* speech may be preceded by a simple bradylalia.

Apoplectiform Attacks.—In a small number of cases attacks of unconsciousness may occur, leaving a unilateral paralysis. This generally disappears in a few hours or days. The symptoms resulting from the stroke, however, may remain, or slowly disappear. The attack, which may also resemble an epileptic one, is accompanied by increase of temperature.

The above are the most important symptoms of multiple sclerosis. Others may also be present. Headache and vertigo may occur. The latter is generally so intense and abrupt in onset that the patient falls to the ground.

The *intelligence* often suffers. The patient's memory weakens and he becomes apathetic. But delusions, hallucinations, and delirium rarely occur. This is important, as multiple sclerosis has been many times confused with paralytic dementia. Certain observations (Schultze) indicate that both diseases may occur together, but I have never seen it.

Another phenomenon is, as I have shown, wrongly supposed to be due to loss of mental vigor,—namely, *involuntary laughter*. The patient laughs against his will, without being in a happy mood. He may be greatly annoyed by this, the laughter often becoming paroxysmal.

Paralysis of the ocular muscles frequently takes place, generally of the external muscles. Myosis and pupillary differences may occur, pupillary rigidity very rarely. *Ophthalmoplegia* is seldom seen, though I have observed three cases of this kind. The voice is monotonous, breaks easily, and hoarseness may be present. *Paresis of the tensors of the vocal cords* has been noticed in some cases. A tremor of the cords during phonation has likewise been seen at times. The tremor may, as I have shown, also extend to the respiratory muscles, producing a kind of staccato breathing.

The *sensibility* is not greatly impaired; but, according to the observations of Freund and myself, only *slight temporary* disturbances happen. They may be stabile, but generally disappear and reappear intermittently. The most frequent form is a hypesthesia; a hemianesthesia is rare. Brown-Séquard's symptom-complex has been observed in a few cases.

Neuralgic pains are often present. The bladder functions are slightly impaired, and the disturbances disappear and reappear in a manner similar to the sensory disturbances.

We will turn now to the *uncommon* symptoms.

(1) *Muscular Atrophy.*—This occurs without any qualitative alterations in electrical excitability. A complete reaction of degeneration never occurs.

(2) *Ataxia.*—It is often difficult to separate slight degrees of ataxia from the tremor. When, with eyes closed, the trembling increases, ataxia certainly exists. The legs are slowly dragged along, the toes stick to the floor; but the leg is raised more than normal and set down forcibly, the heel first. We sometimes find the ataxia combined with atony, and even with Westphal's symptom. Diagnosis is then difficult.

Initial Stage.—It is always difficult to diagnose a disease in the first stage. This is especially true of multiple sclerosis. It begins with *spinal* or *cerebral* symptoms, rarely with *bulbar* ones. Motor weakness of the lower limbs is generally the first symptom, and the signs of a *spastic spinal paralysis* may exist for months or years without other symptoms. Frequent and more exact observations enable us to detect, sooner or later, cerebral symptoms, especially nystagmus, speech disturbance, vertigo, optic atrophy, involuntary laughter, etc., and thereby establish a diagnosis. The cerebral symptoms may also usher in the disease. I have often observed an optic atrophy or neuritis which preceded the other symptoms a long time,—so long that neither the patient nor the physician associated it with the nervous trouble. In one of my cases a unilateral optic atrophy was the only cerebral symptom for twenty years. Tremor, vomiting, vertigo, etc., may also constitute the initial symptoms.

Course.—It is a chronic disease and may be either progressive or step-like in its course, undergoing remissions or improvement, or coming to a stand-still. It occasionally runs an acute course.

The remissions may last months; then comes the relapse, till, after a number of such attacks, a stabile diseased condition is produced. In many cases we can speak of a progressive disease with regressive phenomena.

The relapses may occur spontaneously or come on after injuries or some intercurrent disease.

Atypical Forms.—The disease may resemble spastic spinal paralysis, not merely in its initial stage, but during its whole course. More often we find a combination of *spastic spinal paralysis* with *partial optic atrophy.* I have seen and described a case of *the hemiparetic form of multiple sclerosis,* which has also been written about by Charcot, Edwards, Bouicli, Bickeles, etc.

Bulbar symptoms are occasionally very pronounced; *glycosuria, tachycardia, attacks of asphyxia,* have also been observed in some cases. Lingual hemiatrophy was noticed several times. Multiple sclerosis may be confused with amyotrophic lateral sclerosis, or may be hidden beneath a transverse myelitis.

I have recently observed a disease in young adults which I was forced to consider an acute inflammatory affection of the upper cervical and lower medullar region, inasmuch as there developed an *acute ataxia of the upper extremities,* with bulbar symptoms, a hemiparesis cruciata or alternans, with corresponding alternating extension of the sensory disorder to the one side of the face and to the opposite side of the body. Many of the phenomena quickly passed away. My opinion that these were cases of multiple sclerosis in the initial stage was confirmed in two of them by their further course.

Multiple sclerosis may develop from a post-infectious, disseminated myelo-encephalitis.

Apoplectic attacks with transient hemiplegia resulting from them may occur in multiple sclerosis. Other symptoms and symptom-groups may also develop in this apoplectiform manner. The patient suddenly falls to the ground unconscious or with marked vertigo. After this attack a paraplegia or a paralysis of all four extremities follows, which slowly disappears. These attacks may be repeated. Recurring attacks of hemianesthesia also characterized one case. In one case of insular sclerosis I observed paralysis of the facial, acoustic, and trigeminal, which passed away in a few weeks. Some months afterwards a hemiataxia developed, which also disappeared.

Complications.—It has often been found combined with hysteria. Westphal has noticed it in connection with tabes dorsalis.

Differential Diagnosis.—It can easily be distinguished from paralysis agitans. It has certain symptoms, as tremor, speech disturbance, apoplectic attack, spastic paresis, in common with paralytic dementia. But in the latter the tremor is irregular, not intentional, and may exist when the patient is at rest. The speech is not syllabic, but is more like stuttering; the contractions and tremor of the muscles of the lips produce a peculiar tremulousness of the speech, which is not observed in multiple sclerosis. The psychical symptoms occur from the beginning, while in multiple sclerosis they may even be slight in the last stages.

If the disease begins with cerebral symptoms, it may be confused with cerebral tumor, encephalitis, or simple apoplexy. The head is not painful to pressure, as it is in tumors; and if optic neuritis develops, it rarely reaches the stage of choked disk, and is generally transient, passing away quickly, or it produces a partial atrophy, and is confined to one eye. The constant headache, vomiting, stupor, and retardation of the pulse of cerebral tumor are also not noticed. The tremor that is sometimes seen in cases of tumor has rapid oscillations and is not intentional. Only in cerebellar tumors does it resemble that of multiple sclerosis. In tumors, the symptoms as a rule increase gradatim, in sclerosis with remissions and exacerbations.

There is one type of multiple sclerosis which commences like an acute encephalitis, with focal symptoms. We must wait for further symptoms before a diagnosis can be made. The symptoms of alternating hemiplegia or hemiplegia cruciata may also occur. Some cases have been reported (Strümpell, Bickeles) in which headache, vertigo, and apoplectic or epileptiform seizures, followed by hemiplegia, were the only symptoms of the sclerosis.

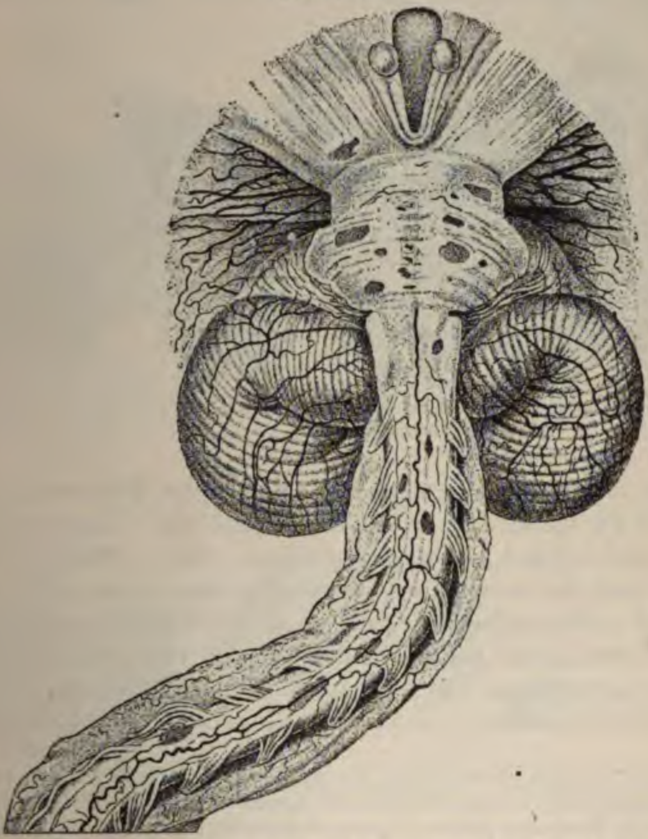
If an apoplectiform attack opens the scene, a positive diagnosis can rarely be made at the beginning. It is, however, always very suspicious if young persons who have not suffered with hypertrophied heart, or syphilis, or alcoholism have an apoplectic attack with transient symptoms of paralysis. There is great probability that encephalitis, beginning sclerosis, or paralytic dementia is present; but further observation only will make an absolute diagnosis possible.

It may also be confused with cases of spinal or cerebrospinal lues, or with combined disease of the posterior and lateral cords. The diagnostic criteria will be discussed under these captions.

There is a general vascular disease of the central nervous system—probably of toxic origin—which resembles multiple sclerosis, but whose basis is not sclerotic foci, but multiple cerebrospinal areas which have softened and produced secondary degeneration of the tracts in the cord and brain (own observation).

Westphal showed that there is a general neurosis which cannot be distinguished from multiple sclerosis either in course or symptoms; he called it *pseudo-sclerosis;* and Charcot's school classed this neurosis of Westphal's as a form of hysteria, without any reason for doing so. Westphal's cases were characterized chiefly by the early appearance of psychic disturbances, apathy, delirium, etc., by slowness of the facial and ocular movements, while nystagmus was absent, and by the paradoxical phenomenon, though no tremor of the foot was present. Westphal himself, however, claimed that the condition of the optic nerve in doubtful cases can make the diagnosis, as a disease of it cannot be expected to occur in this neurosis. Concerning the differential diagnosis between multiple sclerosis and hysteria, see the chapter on Hysteria.

FIG. 131.

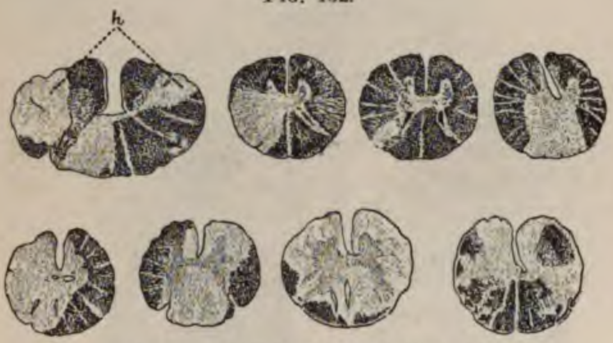


Disseminated sclerosis of the brain and the spinal cord. (Partly schematic.) (After Leyden.)

A mercurial tremor is not intentional, and it may occur at rest. As nystagmus occurs in so many diseases, a diagnosis cannot be made from it alone. A type of pseudo-sclerosis—curable with quinine—occurs in malarial subjects.

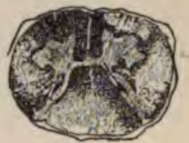
Pathological Anatomy.—The basis for this disease is sclerotic foci which are found throughout the nervous system. They may be generally seen with the naked eye (Fig. 131), as they attain the size of a pea, or even embrace the whole section, and their grayish-blue color is often seen glimmering through the pia mater. (Figs. 132 and 133.) They are found always in numbers, so that we see them scattered everywhere in the spinal cord, with a few in the brain. They are generally sharply outlined, and may be of any shape. They possess a harder consistency than the surrounding tissue. The neighboring tissue is often indurated, so that we find a diffuse sclerosis in addition to the disseminated. Whole regions, as the pons and oblongata, may be atrophied *in toto.*[1]

FIG. 132.



Sclerotic foci at different heights of the cord. *h*, focus. (Weigert-Pal's stain.)

FIG. 133.



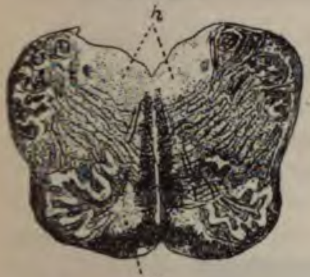
Sclerotic foci in the spinal cord. (Weigert's stain.)

Histologically, we find: degeneration of the medullary substance, *although many axis-cylinders may remain normal;* increase of the connective tissue, which becomes fibrous; and often a vascular proliferation. We are inclined to think that the sclerosis arises from an *inflammatory process commencing in the arteries*, though this assumption is not always supported by the microscopical pictures. Secondary degeneration is rarely seen. Some believe that the disease is due to a primary proliferation of the neuroglia (sclero-gliosis). It is questionable whether a regeneration of axis-cylinders occurs. The ganglion-cells show great resistance to the sclerotic process.

---

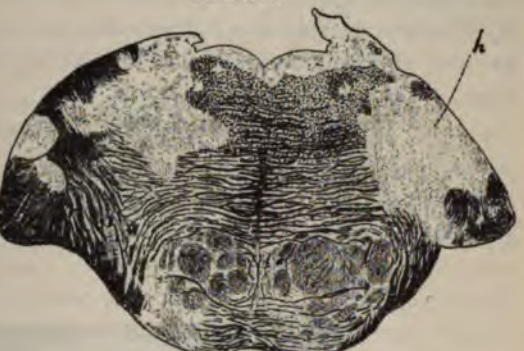
[1] Under the name *diffuse cerebral sclerosis* are embraced many processes which have nothing to do with multiple sclerosis. To these belong anatomical alterations which are similar to the clinical picture of paralytic dementia; also the form of atrophy and induration of a cerebral lobe or of a hemisphere with the symptoms of infantile spastic hemiplegia. Also certain types of idiocy have been referred to a "tuberous" sclerosis of the cerebral cortex (Bourneville). The literature contains a number of observations (Schmaus, Bullard, Heubner, Strümpell), based upon which Heubner has recently been able to set up a clinical picture of diffuse sclerosis of the brain (and spinal cord). The affection occurred mostly with children, and is characterized by paralytic symptoms and psychical disturbances. A *spastic paresis* of the leg develops, extends soon to the arms; apathy and dementia present themselves, passing to idiocy. General paralysis is the final result. Anatomically, a diffuse sclerosis is present, particularly of the white cerebral matter and of the corpus callosum. These conditions must be differentiated from those of multiple sclerosis.

FIG. 134.



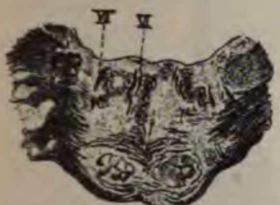
Sclerotic foci in the nuclei and roots of the hypoglossal. *h*, nucleus.

FIG. 135.



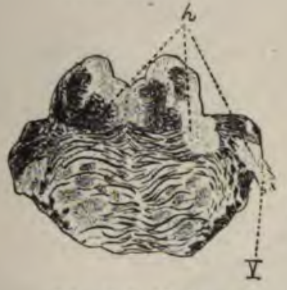
Sclerotic patches in the pons.

FIG. 136.



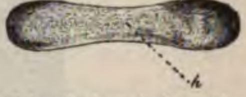
Numerous sclerotic foci in the pons at the height of the abducens and facial roots.

FIG. 137.



Sclerotic foci in the pons; one at the place of exit of the trigeminal.

FIG. 138.



Sclerotic focus in the optic chiasm, frontal section.

Prognosis.—The prognosis is not a bad one *quoad vitam*. The disease generally extends over a long period, though it may cause sudden death, especially *if the medulla* becomes involved. Recovery, however, occurs rarely. Remissions sometimes occur which simulate recovery.

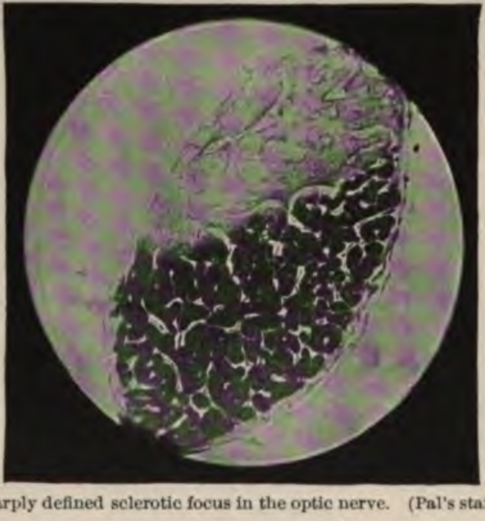
I treated a case of multiple sclerosis in which all symptoms disappeared after a facial erysipelas came on. I saw the disease progress with exceeding rapidity in a vegetarian, who during the spastic paretic stage insisted upon taking a Kneipp cure. The type occurring in early childhood does not seem to be progressive.

Treatment.—Avoid exertion, hot baths, and electricity. Rest is of benefit. Nitrate of silver and potassium iodide may be tried. Marie hopes that the future may produce a real antitoxin treatment. A mild water-cure in Oeynhausen or Nauheim, under careful treatment, may sometimes do good. In apoplectic cases, or those resembling an acute myelitis and encephalitis, a diaphoretic and antiphlogistic treatment may produce excellent results. Local leeching has been a benefit in such cases.

ABSCESS OF THE SPINAL CORD.

This is a very rare disease. In the cases reported,[1] the disease resulted either from traumatism or from metastatic processes,—following a putrid bronchitis, gonorrhea, or abscess of the prostate.

FIG. 139.



Sharply defined sclerotic focus in the optic nerve. (Pal's stain.)

In most of the recorded cases the abscess had its chief seat in the gray substance and was associated with purulent meningitis; in some cases cerebral accumulations of pus developed at the same time. In most cases the upper section of the spinal cord was especially involved; in one case the lower, especially the conus, was purulent (Schlesinger). Generally, the paralysis develops only some days after the signs of meningeal irritation have been present. The symptoms of a diffuse spinal-cord disease then follow rapidly,—paraplegia, anesthesia, sphincter paralysis, etc. In a case of Homén's, in addition to the paralysis of the legs, weakness, tremor, and incoördination of the arms were observed. Fever, subnormal temperature, chills, debility, and other signs caused by the putrid infection are also noticed. Only, however, when the abscess is discovered can an absolute diagnosis be made. Death generally results in a few days.

---

[1] By Jaccoud, Ollivier, Demme, Nothnagel, Ullmann, Eisenlohr, Homén, and Schlesinger.

HEMATOMYELIA, APOPLEXIA MEDULLÆ SPINALIS.

Hematomyelia is as rare as cerebral hemorrhage is common.

Secondary hemorrhages following myelitis, tumors, etc., have no diagnostic interest. The capillary hemorrhages occurring in diseases accompanied by severe tonic or clonic spasms have also no interest for us.

The most important cause of primary hemorrhage of the spinal cord is *trauma*. Probably nine-tenths of the cases are due to this. We disregard here all those injuries in which the bleeding is only an accidental factor; although in these cases, too, a central hematomyelia may extend from the traumatic focus (Minor).

We have in question, however, those hemorrhages of the spinal cord resulting from a fall on the back, feet, or gluteal region, a blow on the back, a forcible inclination of the head forward (Thorburn, Kocher), etc., without the vertebral column or meninges being injured. It has even been caused by the *lifting of a heavy burden*, by military exercises, even by coitus. It is difficult to say whether a predisposition exists,—an abnormal brittleness of the arteries or congestion of the spinal vessels. Spinal hemorrhage is influenced by the existence of a *hemorrhagic diathesis;* as in a case observed by me, in which, in *a bleeder*, the simple attempt to break a piece of wood with his foot produced the symptoms of a spinal hemorrhage. In another case the execution of the ordinary manual exercises with the gun by a soldier produced the signs of a spinal hemorrhage, though there was no tendency to bleeding.

Steffen describes hematomyelia as seen in *purpura;* Teichmüller, in pernicious anemia. *Suppression of the menses, hemorrhoidal bleeding*, etc., are said to produce a hematomyelia. It has been brought on in the puerperium also.

Difficult labor (dystocia) may also produce hematomyelia in the newborn (Litzmann, Schultze, Pfeiffer, Raymond, Oppenheim).

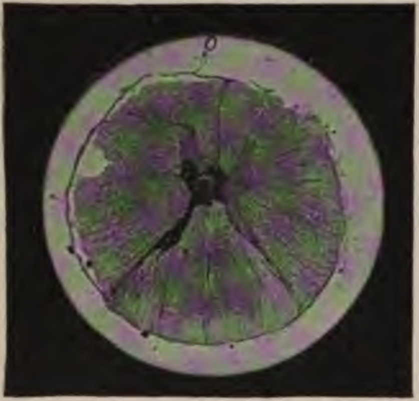
Alcoholism seems to increase the predisposition to hematomyelia (Jestkoff).

The spinal hemorrhage almost always affects the *gray matter* (Fig. 140), though occasionally the crown of the posterior tracts is involved.

Every segment of the cord may be acted upon, but the prominences are especially liable (quantitative excess of gray matter in these regions). The hemorrhage extends over the whole central gray axis or confines itself to one side or part of a side (Fig. 140). It has an especial predilection to stretch in a longitudinal direction in the form of a long tube (*tubular bleeding*). The tissue which is involved becomes friable and softened, and a myelitis often follows the bleeding.

Symptomatology.—The hemorrhage produces a sudden break of continuity in the spinal cord, which happens without warning. The patient sinks to the ground, he is paraplegic, can move none of his limbs, or only his arms; at the same time anesthesia, paralysis of the sphincters, etc., occur. Vasomotor and trophic disorders may soon manifest themselves. Sometimes the paralytic symptoms increase and extend within a few hours.

FIG. 140.



Cross-section of the spinal cord in hematomyelia. Hemorrhage into the gray matter of the left anterior and posterior horns. (After a Minor's specimen in my collection.)

The clinical picture varies according to the region affected by the hemorrhage, and the symptoms generally point to a lesion involving entirely, or almost entirely, the gray matter (*central hematomyelia*). The lumbar prominence is most often affected. We find in such cases a flaccid paralysis of the legs, with loss of reflexes, etc., and, later, signs of muscular degeneration. When the cervical prominence is the seat of the lesion, we find paralysis with atrophy of the upper limbs, spastic paralysis of the lower limbs, etc.; the first being a partial one, the nerve-areas which are affected varying according to the spinal segment involved. Oculo-pupillary symptoms are present whenever the lower portion of the cervical prominence is implicated.

When the hemorrhage confines itself to one side, we have an *acute Brown-Séquard's unilateral paralysis* resulting. The picture is somewhat characteristic, on account of the special implication of the gray matter. In certain cases of Minor, for example, the following symptoms were found: *partial paralysis with atrophy of one arm, spastic paralysis of the leg of the same side, partial loss of feeling,—analgesia and thermanesthesia of the opposite side.* This indicates a hemorrhage in the anterior and posterior horns of one side of the cervical prominence.

The localization and character of the sensory disturbance may also resemble those found in syringomyelia.

A zone of partial loss of sensation is often observed above the limit of the total anesthesia (Minor).

If death does not ensue, and, in uncomplicated cases, this is not to be expected, *improvement* is noticed after some days or the first week. This improvement advances only to a certain stage; a certain number of the symptoms, caused by the damage to the substance of the cord, remain and become chronic. In the beginning, pains in the back are often present, which, however, are rarely severe or lasting. In the first few days or weeks there is a slight elevation of temperature.

The prognosis is good as to life. In extensive hemorrhage death may occur in a few days, or may result later from decubitus, cystitis, etc. Generally, however, improvement and, occasionally, recovery happen. Those symptoms which afier some months are still present as marked as at the onset—especially muscular atrophy with reaction of degeneration—will always remain. The prognosis is also made worse by the fact that the hemorrhage—especially that which takes place in the gray matter of the cervical cord—may start the development of a gliosis.

Minor has shown that traumatic spinal hemorrhage may be combined with the formation of cavities; Van Gieson calls this condition hematomyeloporosis, differentiating it from syringomyelia.

Differential Diagnosis. — Hemorrhage of the spinal membranes causes symptoms of irritation,—severe pains in the back radiating along the nerve-tracts, stiffness of the back, and spinal-root symptoms. Lumbar puncture may clear up the diagnosis (Kilian, Jacoby). It is, however, a questionable procedure in cases of hematomyelia.

Myelitis never occurs so suddenly, although some cases (Williamson, Strull) have been reported which developed within a few hours. It is generally preceded by prodromes, and in most acute cases is accompanied by marked elevation of temperature. In doubtful cases of spontaneous origin it is more liable to be myelitis than hematomyelia.

Acute poliomyelitis is usually preceded by a fever, and its symptoms ordinarily are indicative of disease of the anterior horns.

Treatment.—Absolute rest is imperatively necessary for the first few days, and advisable for the first two or three weeks.

The patient should be put to bed carefully and should lie on his abdomen or on his side. In robust persons *bloodletting* is advisable. *Injections of ergot* are recommended. The patient should avoid coughing, sneezing, etc. Guard against decubitus and cystitis. After the acute stage, warm baths are advisable. Electricity should be used for the atrophic paralysis.

DISEASES OF THE SPINAL CORD RESULTING FROM PRONOUNCED ALTERATIONS IN ATMOSPHERIC PRESSURE (DIVER'S DISEASE, CAISSON DISEASE).

These occur in divers, harbor- and bridge-workers, who are forced to work under water in caissons,—*i.e.*, divers' apparatus,—in an atmospheric pressure of from one to four or five atmospheres. Cerebral or spinal paralysis may occur in a workman soon after quitting the apparatus, from sudden and pronounced alteration of the density of the surrounding air. Upon leaving the caisson he complains of a feeling of weight in his head, vertigo, *buzzing in his ears, malaise*,[1] and weakness in the legs. The symptoms, which may increase within a few minutes to paraplegia, generally represent a diffuse disease of the dorsal cord: spastic paralysis, anesthesia, weakness of the bladder, etc., are found. Severe pains are also sometimes noticed. In a case which I observed for a long time the *paralysis* was accompanied by marked rigidity of the legs and of the abdominal muscles; and every attempt at movement produced clonic contractions of the latter, involuntary passage of the urine, and erections of the penis.

If the symptoms of paralysis are slight, a complete *restitutio ad integrum* is possible. Death, however, often results, the severe cases being incurable.

Few post-mortem examinations are on record (Leyden, Schultze, Rensselaer, Catsaras, Hoche). Leyden found slight fissures and ruptures of the dorsal cord. Local necrobiosis in small patches was observed in one case. It is thought (Leyden, Bert, Hoche) that bubbles of gas escape from the blood through the sudden decrease in the atmospheric pressure and produce an air-embolus of the small arteries of the spinal cord, particularly in the lateral and posterior tracts, with secondary softening. Other theories that have been advanced need not be discussed here. The treatment is the same as in myelitis. Divers should be warned of their danger and cautioned about returning to the normal atmospheric pressure too quickly. So far as I know, this is always done.

TUMORS OF THE SPINAL CORD.

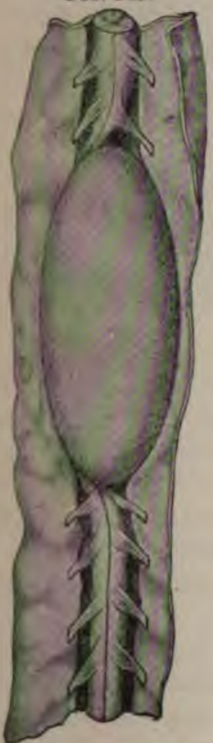
Excluding gliosis and glioma, which will be discussed in a separate chapter, we can say of the remaining spinal tumors that they mostly arise from the meninges. *Lipoma* and *echinococci* develop externally to the dura. The tumors springing from the roots may also be extradural. *Internally* to the dura we have myxoma, sarcoma, psammoma, syphiloma, tubercle, and fibroma. The sarcoma forms multiple tumors, though it may extend diffusely over the whole surface of the meninges. Cases have been described in which to a great extent the spinal cord was replaced by the tumor (Ross, Malacaster, Forster). Multiple neuromata and sarcomata may form in the spinal roots and appear at the same time in the brain, spinal cord, and cranial nerves. Echinococci and cysticerci have also been observed. Echinococci lie generally between the dura and the bones; after the latter have been worn away, they may appear on the surface. Glioma and tubercle often start in the gray matter of the cord; sarcoma also may originate there (Schiff).

---

[1] Friedrich and Tausk have carefully studied this symptom, and have found alterations in internal organs (dilatation of the heart, etc.).

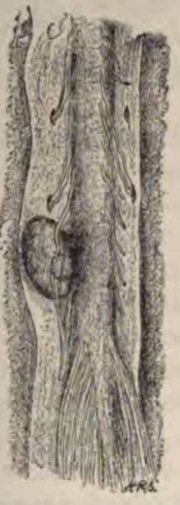
Lipomata are congenital. Heredity seems to play a part in the etiology of multiple tumors. Excluding the infectious and parasitic tumors, nothing positive is known of the etiology. The frequency with which the symptoms appear after traumata is noteworthy. The tumors arising from the membranes of the cord are usually small, but may grow considerably in a vertical direction and follow the cord for two inches or more. (Fig. 141.) They may also extend along the periphery so as to sheathe the cord like a tube.

FIG. 141.



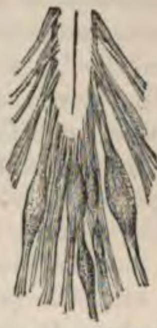
Tumor of the medulla spinalis. (After Braubach.)

FIG. 142.



Tumor arising in the dura of the upper part of the lumbar prominence. (After Gowers.)

FIG. 143.

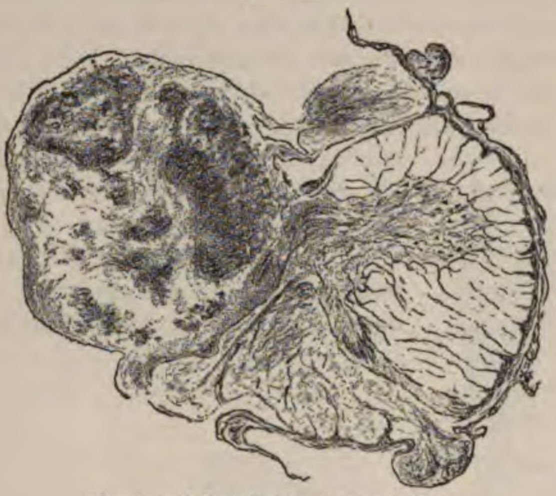


Neuroma of the cauda equina. (After Lanceraux.)

Most of these tumors (excluding the syphilomata) have a slow growth. They involve the neighboring roots and press upon the cord until it is bound down and reduced possibly to one-fourth of its previous size, while the tumor to a certain extent forms a nest in it and lies imbedded as in a niche of the spinal gray matter. Histological alterations are often very slight; at other times inflammatory and softening processes occur around the tumor; secondary alterations also take place.

The tumor rarely absorbs the substance of the cord. (Fig. 144.) The tumors lying externally to the dura exist a long time before they disturb the functions of the cord through pressure. Here have been found gliomata, sarcomata, fibrosarcomata, and in one case an extradural lymphangioma.

FIG. 144.



Tumor medullæ spinalis. (Cross-section.)

The tumor may develop in any part of the cord, but oftenest in the dorsal region. The cauda equina is also often affected. (Fig. 143.)

Symptomatology.—In most of the old observations the tumor was recognized only on post-mortem examination.

The clinical interest in the disease, however, has greatly increased since the noted case of Gowers and Horsley, who showed that the tumors were accessible to operative treatment, and since then the symptomatology has been made more exact and complete.

Spinal-root symptoms are the first signs of the trouble, and, as at first only one root or a pair of roots have been affected by the tumor, we find that *neuralgic pains* in a definite nerve area, particularly unilateral or bilateral *intercostal neuralgia*, are the earliest symptoms.

These pains are slight at first, increase gradually until they become as "sharp as a knife-edge," next become paroxysmal, and finally only a continued dull pain is present, which undergoes exacerbations. It rarely happens that they are entirely absent.

Hyperesthesia may at times accompany these pains. This *neuralgic stage* may last for months or years before further symptoms are noticeable. If the tumor has its seat on an anterior root of the cervical prominence or in the lumbo-sacral region, we next obtain motor symptoms of irritation in a definite muscular region,—tremor, contraction, and spasms.[1] These are often hidden by reason of the development of a degenerative paralysis in the corresponding muscles. In reviewing the existing observations, it is evident that symptoms indicating irritation of the posterior roots are the most prominent, even in those cases where the tumor lies equally in the anterior and posterior parts.

In compression of the anterior roots also, paresis may exist a long time before alterations in electrical excitability occur (Bruns, Oppenheim).

Anesthesia may develop later on, or the *neuralgic or anesthetic zones may increase in extent upward or downward*, or symptoms of compression of the cord may occur. Symptoms of meningeal irritation, as pain or stiffness in the back, may appear, though the latter is never pronounced. The compression of the spinal cord produces, when it occurs above the lumbar cord, *spastic paralysis;* and the *muscular rigidity* and *increase in reflex excitability* are in no other disease so marked.

The tumor, as a rule, is unilateral at first. The paralysis involves, therefore, only one leg at the beginning, though the reflexes are generally increased on both sides.

The spinal hemiparesis, however, soon changes to a paraparesis or a paraplegia, and the muscular rigidity increases to such a degree that marked flexion contractures in the thigh and knee develop.

The sensibility in the legs may remain unimpaired for a long time. More often, however, there appears, in the stage where the motor weakness affects entirely or principally one leg, a hypesthesia or anesthesia of the other extremity,—*i.e.*, the symptom-complex of Brown-Séquard's *unilateral paralysis* often occurs when the tumor implicates the cord above the lumbar region. It is never marked, and soon disappears, the paralysis and anesthesia gradually involving both sides.

The *functions of the bladder and rectum* are often affected early in the disease. At first the patient complains merely of an increased, imperative desire for urinating; later, incontinence of the urine and paradoxical ischuria develop.

---

[1] General tonic spasms with opisthotonus, etc., were noticed several times.

Modifications of the symptoms occur according to the region influenced. Compression of the lumbar cord causes paralysis with atrophy, the spinal symptoms resembling a lumbar myelitis. The severe pains in the nerve-tracts from the lumbo-sacral plexus furnish an important differential point. Cervical tumors cause spinal hemiparaplegia at first, later a paraplegia of all four extremities, the paralysis of the upper being spastic or atrophic or a combination of both.

In *tumors of the cauda equina* the pains occur in the pelvic region, radiating to the anal and perineal region and along the sciatic nerves. In one case the paralysis confined itself to the bladder; in another a degenerative paralysis in the area of the ischiadic plexus appeared. Tumors that involve the posterior tracts may produce ataxia; if they occur at the height of the upper lumbar cord, the knee reflexes may disappear early.

In cases of multiple formation the symptoms indicate more than one lesion. The presence of tumors in other regions is an important aid to the diagnosis. The course is chronic and may extend over years. Spontaneous remissions often happen.

Treatment.—If syphilis is suspected, a thorough antisyphilitic treatment must be instituted. In non-specific cases the physician formerly could do little more than attempt to still the pains, but it has in recent years been shown that operative measures may produce complete recovery. Although recommended by Erb in 1878, it was Horsley and Gowers's case of 1887 which showed what could be done in this way.

Gowers and Horsley believe that almost all the intradural tumors are operable. We have now eighteen recorded cases of surgical interference for spinal tumors. Recovery was noted in five; in most of the other cases death followed immediately after the operation. Even though the results have not been very good, we must remember that without operation the patient will surely die, while he *may* be cured by surgical interference. Multiple tumors which spread diffusely over the surface of the spinal cord and those which arise directly from it are not operable. Metastatic tumors also had better be left alone.

An absolute diagnosis is not the only thing that must be made before the exploratory operation, but the tumor must be *localized.* This causes the most difficulty, as in many cases the tumor is sought for too low down. As Bruns has remarked, it is generally only possible to determine the segmental height of the upper border of the tumor. It is necessary to search for the highest seat of sensory, paralytic, and painful symptoms and to refer them to the highest segment of the cord which could be in question, and finally to ascertain that dorsal spine which corresponds to the upper segment. Gunshot wounds are especially difficult to locate. The use of Röntgen rays is advisable for this purpose, and the examiner must keep in mind the facts that have been given in previous pages concerning localization.

SPINAL GLIOSIS AND SYRINGOMYELIA.

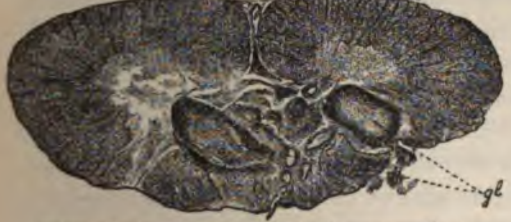
These two pathological conditions will be considered together, because they are generally associated with each other and cannot be clincially separated.

We understand by *spinal gliosis* a neoplastic process in the gray matter of the spinal cord which does not, as a rule, produce much of an increase in the size of the cord. Degeneration of the neoplasm, especially of its central parts, causes cavities (syringomyelia).

Hydromyelia, a congenital dilatation of the spinal canal corresponding to hydrocephalus, has been known a long time (Ollivier, Lancereaux) without having had any clinical importance. Later, Hallopeau and Joffroy believed that a central myelitis of the gray matter was responsible for the cavities. Still later, it was learned that cavities may result from *degeneration of tumors* (Westphal, Simon). At first this process had only a pathologico-anatomic interest. In the text-books of the past two decades, syringomyelia has been discussed under the rubric of rare and curious diseases. With the observations of Kahler, Schultze, etc., this disorder began to be studied clinically. It did not take long, however, for them to complete the symptom-complex; so that to-day it is not difficult to diagnose this disease in life. (Hoffmann, Schlesinger.)

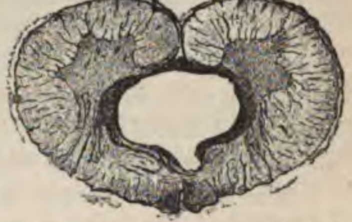
Pathological Anatomy.—The spinal cord may appear swollen, especially in the lower cervical region, where we can detect fluctuation; and often palpation is sufficient to show us that the cord has been changed into a tube. Upon section, the cavity is the first thing noticed. It may be so large that the tip of the little finger can be placed in it; but is generally small, and at first glance looks like a dilated spinal canal. In many cases we do not notice any cavity, but a tumor (Figs. 145 to 148), which may extend through the cervical and dorsal region,

FIG. 145.



Spinal gliosis. (After a Westphal's specimen in my collection.) (Carmine stain.)

FIG. 146.



Syringomyelia. (After a Westphal's specimen in my collection.)

and even into the medulla. As a rule, a cavity is seen in the tumor at some place in the cord. The neoplasm may be confined to one side or to one of the posterior horns.

Microscopical examination reveals the fact that the tumor consists of glia cells and glia fibres. This gliomatous tissue varies considerably in the amount of each that it contains. The cavities are encircled by a hard, dense membrane, which may be covered with epithelium.

Congenital developmental anomalies are the basis of the process. These may affect the central canal, or nests of glia cells are left in the region of the central canal to be irritated spontaneously or through trauma, and which proliferate, producing neoplasms (Hoffmann). The central canal is found in the centre of the tumor, if the process began in the cells in its vicinity. A cavity may be caused by a degeneration of the neoplasm, or there may have been two canals from the first. According to whether the canal is primary or secondary do we find it surrounded by epithelium or not. Other processes, as, for instance, a circumscribed meningeal disease, may lead to the formation of cavities. But it is doubtful whether on account of this a division into different forms (Marinesco) is practicable.

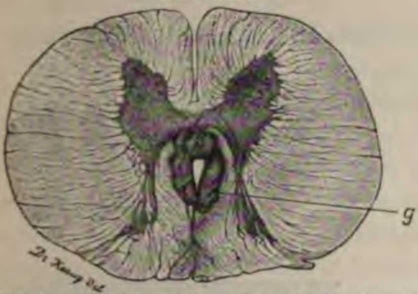
Causes.—It has repeatedly been noticed that the disease follows a trauma. It is probable that a congenital predisposition exists, and the trauma then excites the cellular proliferation. Experimental observations (Schmaus) also indicate this method of development. According to Minor, a central hematomyelia produced by the trauma often starts the process. Spinal hemorrhage caused by congenital injuries may also come into action (Schultze). A splinter of bone entering the spinal cord produced a syringomyelia in one case. Cavities have often been observed in *syphilitic* individuals and accompanying other diseases of the cord.

Symptomatology.—In the typical cases, in which the gliosis involves especially the cervical swelling, we find the following triad of symptoms: (1) *Progressive muscular atrophy* of the upper extremities; (2) *partial loss of sensation* in the upper extremities, neck, and trunk; (3) *vasomotor and trophic disturbances.*

The muscular atrophy begins mostly in the hands, more rarely in some other section of the upper limbs, and shoulder-muscles. The small muscles of the hand are first affected; the interosseous spaces sink in; the balls of the thumb and small finger emaciate; a "claw-hand" develops, and the whole picture strongly resembles the spinal form of progressive muscular atrophy. In former years this disease was often confused with gliosis. The muscles involved in the atrophy show, as a rule, a fibrillary tremor. The electrical examination reveals a reaction of degeneration, which, however, appears in only a few muscles or muscular regions, and is often only a simple quantitative decrease of the excitability. In some cases with an insidious course, notwithstanding the marked atrophy, I did not find any noticeable disorder of electrical excitability.

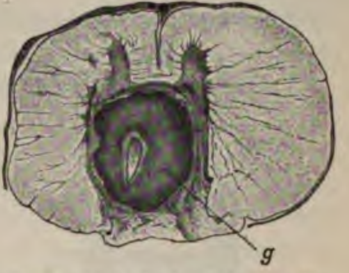
The atrophy is rarely symmetrical. As the atrophic paralysis involves particularly the ulnar and median region, the over-action of the muscles innervated by the musculospiral produces a "preacher's hand." One hand is generally affected more than the other, or only one hand may be attacked.

FIG. 147.



Gliosis and syringomyelia. (Stained with nigrosin.)

FIG. 148.



Gliosis with beginning syringomyelia.

We have a peculiar form of disorder of sensibility. While touch and pressure, and also the sensation of position, are usually intact, the *sensation of pain* is more or less destroyed and the temperature sense also decreased or lost. This partial disorder of sensation is not limited to a single region, but is found over a whole arm or both arms, or the whole upper part or one side of the body. The extent corresponds to the surface innervated by the involved posterior roots or segments. A Brown-Séquard type is occasionally found. Paresthesia is commonly present. Pain is also a not infrequent symptom. The patient at times has no knowledge of the sensory disorder.

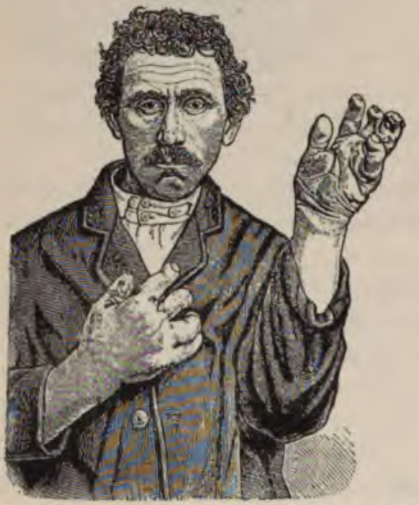
Trophic and Vasomotor Disturbances.—Vesicles often form on the skin (of the hands), which break and leave badly-healing sores. Wounds and scars are often found upon the hands of these patients, due partly to the fact that they do not experience pain or heat or cold, and therefore easily injure themselves without noticing it. The hand and lower arm are often of a blue-red color or reddened; edema is noticed sometimes. Other disturbances observed are fictitious urticaria, pemphigus, phlegmonous processes, panarities, keloids, necrosis of the bones, ankylosis of the joints, thickening of the terminal phalanges, and mutilation of the phalanges. (Fig. 149.)

I found a Dupuytren's fascia-contracture in two cases of gliosis. An acromegaloid hand has also been observed. Marinesco has described a peculiar appearance of the hands as "*main succulente*," and in contradistinction with Dejerine, among others, has considered it pathognomonic. Its chief characteristics are a diffuse swelling, especially on the back of the hand. Marinesco considers it to be a hyperplasia of the subcutaneous connective tissue, but vasomotor disturbances are also factors. The hand is likewise smaller from the muscular atrophy, feels cold and dry, etc.

An abnormal brittleness of the bones is produced by the disease; even *painless, spontaneous fractures* occurring.

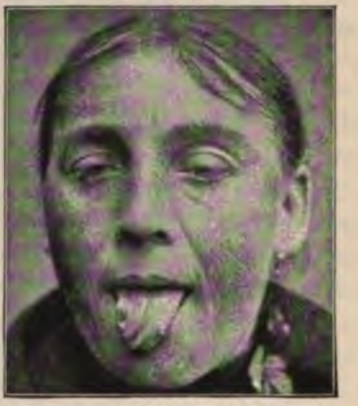
Trophic changes in the joints, closely allied to the *arthropathies* of tabes, are often seen (Bernhardt, Sokoloff, Klemm, Graf, Londe), only the joints of the upper extremity being, however, as a rule, involved. Suppuration of the diseased joint is occasionally noticed.

FIG. 149.



Absence and deformity, etc., of the end phalanges from syringomyelia. (After Hoffmann.)

FIG. 150.



Lingual hemiatrophy in syringomyelia.

The cause of the scoliosis, kyphoscoliosis, thorax en bateau (Marie), etc., which sometimes are present, is not known.

Unilateral hyperidrosis is frequently complained of. The pupils and palpebral fissures of one or both sides are often smaller than normal. The eye, however, reacts to light, though the dilatation usually observed upon shading the eyes is incomplete.

ATYPICAL FORMS AND UNCOMMON SYMPTOMS.—The chief uncommon symptoms are: Bulbar symptoms, especially anesthesia of the trigeminal region; paralysis of the vocal cords and palate, even complete *recurrens paralysis*, has been observed and referred to involvement of the vago-accessorius. Nystagmus is not uncommon, and mellituria has been observed. Lingual hemiatrophy (Fig. 150) occasionally occurs; Chaubanne, Schlesinger, and Dejerine have described hemiatrophia facialis.

The gliosis may confine itself to one side of the spinal cord (Rossolimo, Dejerine, and others). The process may also confine itself to the posterior or anterior horns of one side, the symptoms, likewise, being then limited.

Dejerine had one case in which there was only an atrophy of the shoulder.

I observed three cases in which the disease restricted itself to the lower limbs at the commencement.

Choked disk and optic neuritis were noticed several times; it is doubtful whether they were pure cases of gliosis.

A group of cases in which, in addition to the analgesia and thermoanesthesia, there existed tactile anesthesia and panarities of the fingers, was described by Morvan as a separate disease-complex. Recent observations, however, show that it is merely a form of syringomyelia.

When the posterior tracts are especially involved, the disease, anatomically and clinically, may resemble tabes dorsalis.

Development and Course.—It comes on gradually and insidiously, and generally attacks persons in middle life, though childhood is not exempt. The trophic disturbances are the symptoms first noticed by the patient; and if he goes to a physician, the latter will at that time detect the sensory disorder.

It progresses gradually and may exist for years. I treated a woman, sixty years old, who had had the disease for eighteen years. Remissions may occur. The acute paralysis exhibited in some cases is probably caused by hemorrhage, edema, or serous exudates into the cavities.

Death generally results from cystitis, decubitus, septicemia, or from involvement of the medulla, or through any intercurrent disease.

Differential Diagnosis.—For the signs which distinguish it from progressive muscular atrophy of spinal origin and amyotrophic lateral sclerosis, see the descriptions of those diseases. As long as sensory disturbances are absent, a positive diagnosis is impossible. Even then, however, the flaccid atrophic paralysis in syringomyelia, without an increased knee-jerk, enables us to distinguish it from amyotrophic lateral sclerosis.

The gliosis may resemble a spastic spinal paralysis, but the partial sensory disturbance is rarely absent continuously.

Caries of the spinal cord may present a similar clinical picture,—progressive muscular atrophy of the upper extremities, especially the hands, sensory disorders, perhaps also similar pupillary changes. But the symptoms of tuberculosis and vertebral disease are seldom missing, and the sensory disorder is scarcely ever of a partial character.

In diseases of the brachial plexus, be they of traumatic, toxic, or infectious origin, symptoms of irritation are the most prominent phenomena observed. The anesthesia is generally complete, and its area corresponds to the nerves that are involved.

In the so-called vasomotor neuroses (acroparesthesia) objective symptoms are absent, or only slight tactile hyperesthesia of the finger-pulps is found.

Hysteria may simulate syringomyelia, but the distinguishing symptoms of each should prevent any difficulty in diagnosis. Hematomyelia may sometimes resemble gliosis, though the former is always a retrogressive disease. In doubtful cases the further course must show whether a gliosis has developed from the hematomyelia.

Hoffmann wishes to distinguish gliosis from glioma of the spinal cord. This is an acute or subacute affection and runs a much more rapid course. The symptoms also more nearly resemble a transverse myelitis.

Morvan's type of syringomyelia is difficult to distinguish from leprosy, especially the macular form of it. They resemble each other so much that Zambaco and Marestang have endeavored to make syringomyelia a form of leprosy. This is, however, impossible, as in the bodies of lepers the anatomic process of gliosis has never been found, and in the spinal cords of those who died from gliosis the lepra bacillus has never been detected.

In leprosy the cutaneous lesion extends in multiple circumscribed plaques on the lower limbs and face, while in syringomyelia they are generally confined to the upper extremity. The facial paralysis of leprosy, the trophic disorders of the face, the pigmented or pigmentless spots, the leprous knots, are all absent in syringomyelia. The sensory disorder of leprosy is not dissociated and partial, as in syringomyelia, and generally appears in islets or corresponds to the innervation of peripheral nerves. The muscular atrophy of leprosy begins at the distal parts of the extremities, while in gliosis it may commence in the shoulder-girdle. Paralytic symptoms of a spastic nature in the legs, bulbar symptoms, nystagmus, etc., favor a diagnosis of gliosis. The detection of the bacillus of leprosy makes the diagnosis certain.

Raynaud's disease and allied tropho-neuroses are sometimes confused with syringomyelia, but they should be easily distinguished.

The prognosis *quoad sanationem* is a bad one. Though generally progressive, it sometimes comes to a stand-still. Remissions are more often observed. In one case, where lues had been present, recovery occurred under the use of potassium iodide.

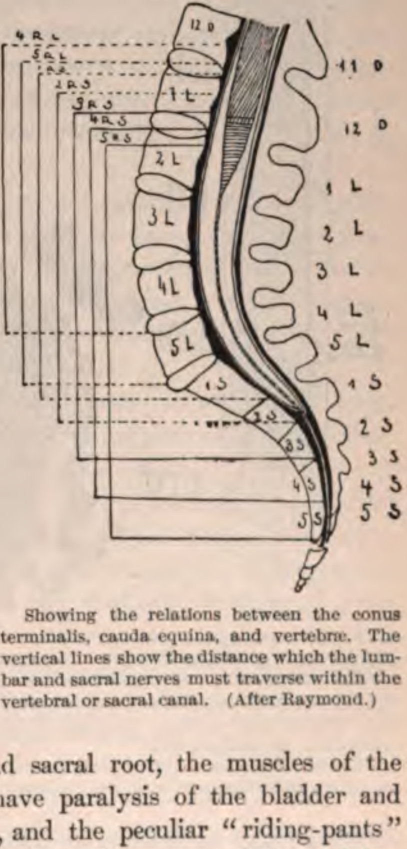
Treatment.—The patient should be cautioned against injuring himself, or using his arms very much. Galvanism may be tried, or a mild faradism of the atrophied muscles. Lumbar puncture has lately been recommended, but why I do not see.

Arsenic and silver nitrate may be given internally.

DISEASES OF THE CAUDA EQUINA (AND FILUM TERMINALE).

The diseases of the cauda equina demand *separate* treatment only by reason of the conglomeration of *nerve-roots* that form it. Disease processes may extend to the cauda from the lumbar vertebræ, the sacrum, the meninges, or develop independently. Injuries, fractures, luxations and their results, traumatic hemorrhages, and puncture-wounds in lumbar punctures are the causes of diseases in this region. Tumors, syphilitic neoplasms, and meningitis may also involve this region. The cauda equina is involved *in toto* when the lesion is high. The lower it is, the less the number of roots that are involved. A complete lesion of the cauda equina produces an atrophic paralysis of the muscles of the lower extremities, an anesthesia of the same, paralysis of the bladder and rectum, and impotence. If the upper lumbar nerves are intact, the ileohypogastric and the ileo-inguinal regions, as well as the testicle, retain their sensibility. If the process is below the third lumbar root, the crural and obturator are not affected. If the disease only involves the region below the second sacral root, the muscles of the lower limbs remain intact, and we have paralysis of the bladder and rectum, and of the sexual functions, and the peculiar "riding-pants" anesthesia described before. This involves the anus and its vicinity, the perineum, the posterior scrotal region, the urethra, the mucous membrane of the bladder, and a strip of skin, innervated by the cutaneus femoris nerve, upon the posterior inner surface of the thigh. The electrical excitability of the sphincter ani, as well as its reflex action, is also lost.

FIG. 151.



Showing the relations between the conus terminalis, cauda equina, and vertebræ. The vertical lines show the distance which the lumbar and sacral nerves must traverse within the vertebral or sacral canal. (After Raymond.)

It need not be further shown how it is that the lower the lesion is, the less the area involved until a disease of the coccygeal nerve produces only a paralysis of the levator ani and an anesthesia of the coccygeal region.

The diseases of the cauda equina produce a symptom-complex similar to that of the diffuse diseases of the lumbosacral cord. An almost complete analogy exists between the diffuse diseases of the filum terminale[1] and the lesions of the lower sacral roots. As first established by myself, disease of the filum terminale produces the following symptoms:

Paralysis of the *bladder and of the rectum*, *anesthesia of the "riding-pants" type, loss of the sexual reflexes, of the Achilles reflex*, etc., with intact motility in the lower extremities. Many others have since described the same symptom-complex; and in those cases that came to a post-mortem examination (Sarbo), myelitis and hematomyelia (Fig. 153), primary hematomyelia, and syphilitic processes were the causes observed.

FIG. 152.

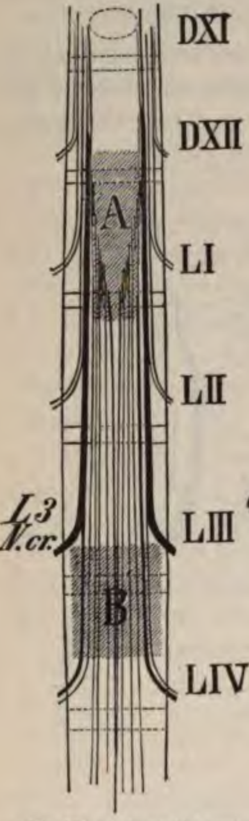


FIG. 153.

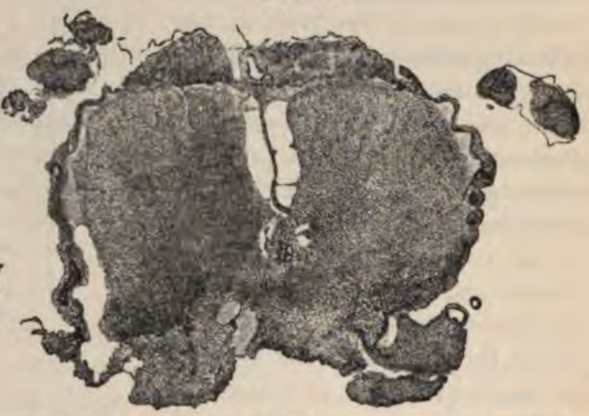


Fig. 152.—Schematic representation of the vertebral column with the lower part of the spinal cord and the cauda equina. DXI–LIV, eleventh dorsal to fourth lumbar root. The third lumbar root, *L 3* (with its crural and obturator fibres), is made more prominent by dark shading. A, lesion at the height of the lower part of the spinal cord; B, lesion at the height of the third lumbar vertebra, involving the cauda equina. The figure is meant to show that both lesions cause about the same symptoms. (After Schultze.)

Fig. 153.—Traumatic myelitis and partial disintegration of the lower part of the spinal cord. (Specimen stained with carmine and alum-hematoxylin.)

---

[1] Raymond's recommendation to consider the filum terminale as that part of the sacral cord which extends from the third sacral nerve downward has been adopted.

It has been a task of many (Schultze, Raymond, etc.) to establish a differential diagnosis between diseases of the filum terminale and of the cauda. The following criteria are of value: (1) The seat of the pain, the local sensitiveness to pressure, and especially the seat of the deformity. If these are on the lower lumbar vertebræ or on the sacrum, it indicates a disease of the cauda. (2) Marked anesthesia, and especially the discovery of *partial loss of sensibility* (analgesia, thermanesthesia), point to a disease of the filum. (3) Diseases of the cauda equina cause almost always *severe pain* in the sacrum, bladder, perineal, anal, and sciatic regions, which are absent or slight in diseases of the filum. (4) Trophic disorders generally develop faster and more often in lesions of the filum. A fibrillary tremor in the atrophic muscles is by some thought to point in doubtful cases to a conus disease. Diseases of the cauda equina are almost always bilateral, though we have no knowledge of unilateral affections of the conus. Traumatic hemorrhages evoke symptoms much earlier when they affect the conus than they do when the sacral canal is involved.

Peripheral injuries to the sacral nerves are generally unilateral, and are often accessible to a direct examination by the rectum or vagina. Tabes dorsalis also may commence in the sacral roots, but there is generally no trouble in diagnosing it. We sometimes have a neuralgia limiting itself to the ano-vesical region (Weir Mitchell), but it has none of the objective symptoms of the caudal disease.

There is a congenital, as well as acquired, paralysis of the sphincter vesicæ, more rarely of the sphincter ani,—I have noticed it in children and adults,—the anatomical basis of which is uncertain.

The prognosis of diseases of the cauda is in general better than that of diseases of the spinal cord. Although they may occasionally be acute and cause death (Schultze), improvement is often noticed, or they come to a stand-still, especially in traumatic and syphilitic diseases of these nerves.

Operative treatment has a comparatively good prognosis, as the cases of Thorburn, Laquer, Shaw, and Busch show. I saw one case of syphilitic origin improve under appropriate treatment.

THE THIRD GROUP

of diseases of the spinal cord consists of *those which have no anatomical foundation.* The forms and conditions which we include under the name of functional neurosis, and which embrace spinal neurasthenia, spinal hysteria (?), spinal irritation, etc., will be sufficiently described later on; and we will, therefore, only say a few words here about "spinal concussion." A fall on the back, a blow, or a general concussion, as in railroad accidents, may produce grave results. Internal hemorrhage may take place in the cord and meninges without there being any external injury; even lacerations of the cord may occur in this way. The symptomatology need not be discussed here. As a result of such concussions a condition of general paralysis may occur which denotes a functional inhibition of the spinal cord, and produces death without anything being revealed at the autopsy. This condition and result of shock has been known a long time, and referred to a molecular concussion or a reflex inhibition of the cord. Kocher, however, denies that shock may produce any results without any lesion of the cord or brain.

Symptoms of Shock.—We find "great prostration; the eyes are sunken, not bright; the look is fixed, staring, and vacant; the extremities are cold; the skin resembles marble; the hands and feet are slightly cyanotic; the temperature is 1° to 1½° C. below normal, and the pulse is hardly perceptible. The sensorium, is, however, free; voluntary movements are possible, but limited and feeble. The sensibility is lowered," etc.

In many cases the symptoms of a nervous trouble come on gradually and insidiously after the injury. Formerly it was thought to be a chronic meningo-myelitis which resulted, and therefore the name *railway spine* was given to the nervous trouble arising after railroad accidents. It is very uncommon for such accidents to produce lesions without any external injury, and such diseases should be grouped for the most part in the category of functional neuroses.

I. CONGENITAL MALFORMATIONS OF THE SPINAL CORD AND ITS ENVELOPES.

Some of these, as amyelia (absence of the spinal cord), atelomyelia (defect of a certain segment from developmental inhibition), diastematomyelia (bifurcation of the cord), diplomyelia (double-cord), cannot exist and the patient live. They have, therefore, no clinical interest. Certain other malformations, as asymmetries and heterotopia, produce slight disturbances of function, but are not of diagnostic importance.

Ira van Gieson showed that the majority of the *alterations* of the spinal cord described as heterotopia were artefacts. Rachischisis, or spina bifida, is, however, of clinical interest.

Embryonal defects result from failure of the spinal canal to close (rachischisis) or from the meninges becoming cystic and forcing themselves through the cleft in the canal (meningocele, etc.). In many cases the division is so complete that the soft membranes of the cord and the spinal cord itself protrude (myelocele).

V. Recklinghausen characterizes as *myelocystocele* a cystic tumor which forms in the spinal cord and produces a cystic, dilated, embryonal medullary tube which pushes through a cleft in the vertebræ. In simple rachischisis the spinal cord remains intact, but extends in this, as in the other types, downward into the sacral canal. The filum terminale is *adherent* at its original situation. In most cases of spina bifida the lower section of the spinal cord is more or less limited in development. The roots of the cauda equina are often degenerated and take an abnormal course, being compelled to extend backward and upward in order to reach their place of exit in the spinal canal.

These tumors, as a rule, are found in the lumbosacral region of the neural canal, in the median line, and have a circumference varying between the size of a nut and the head of a child. They are elastic and fluctuating, and at times the defect in the vertebræ may be felt at the base or side of the tumor. The tumor is sometimes absent, and a cleft only is seen (spina bifida occulta). The skin covering the tumor is normal, or at times thinned and covered with hair. Patients with a spina bifida often present other developmental anomalies. Pressure on the tumor may produce disturbance of the cranial functions, even unconsciousness.

The anatomical *changes* in the lumbosacral cord and in the spinal roots are the phenomena that are of *neurological* interest. They are to a great extent congenital, but may only develop in the adult. That they may develop late in life is true, but it is generally a result of injuries, lacerations, or secondary inflammations.

TALIPES (pes varus or equino-varus paralyticus) follows very often from paralysis of the muscles of the lower leg. In twenty cases De Ruyter found this symptom eight times. A *complete paraplegia* with atrophy of all the muscles of the extremity may also take place. Bladder and rectal disturbance, anesthesia, and ulcerations may also occur. The knee-jerks may be diminished. The persons affected rarely reach an old age, and the more prominent the signs of paralysis are the more their life is in danger.

The *treatment* is *surgical*. Extirpation of the sac is indicated in meningocele and myelocystocele. Hydrocephalus is a contraindication to operation (Broca, Schede). Although some cases of myelomeningocele have been operated upon successfully (Bayer and others), little is to be expected in such cases. Morton's fluid has been injected into the sac with success.

II.

We wish to speak here of certain types of spinal paralysis of which we have a very uncertain pathological anatomical knowledge. To these belong the *reflex paralyses*, which are regarded as cases of pure paraplegia or transverse myelitis, and which often develop as a sequence of diseases of the urinary tract, the rectum, or the uterus. As to most of the cases, we do not consider that they are a result of arterial spasm without an anatomico-pathological basis, as was formerly believed. Even if it has been experimentally shown that strong excitation of the internal organs (for example, contusion or laceration of the kidneys) may cause a paraplegia which soon disappears, we believe that anatomical processes, neuritic, meningitic, or myelitic, are at the bottom of it.

Disease of the mucous membrane of the bladder, of the urethra, of the prostate, etc., may result in paralytic conditions. These may be caused by an inflammation or suppuration along the nerve-tracts which extends to the meninges or to the cord, or by septic matter, or the toxins produced by it, acting directly upon the cord.

The theory of reflex paralysis, however, has not yet been abandoned. We shall return to it later. We will here only refer to cases of phimosis which coexisted with paraparesis of the legs and paralysis of the bladder, and in which operative treatment of the phimosis relieved the other symptoms. I observed one case where catheterization of a patient produced a paraparesis which without doubt was functional.

We must also consider as functional those transient paralyses which occur after severe diarrhea, after the use of drastic purgatives, and from irritation from worms.

Though some cases have been recorded in which, after injury of the peripheral nerve, an *ascending neuritis* ascended to the spinal cord and produced a myelitis, causing a paralysis of spinal type, still most of the central paralyses happening after a peripheral injury are cases of traumatic neuroses. Simple weakness, with difficulty in walking, as it occurs after acute infectious diseases, great loss of blood, etc., without any evidence of a spinal lesion, is probably due merely to malnutrition. Perhaps anemia of the spinal cord may be partially responsible for such conditions. The prognosis is always good. On the other hand, the observations of Lichtheim and others (see page 143), that severe anemia may produce structural changes in the spinal cord, make it difficult to determine whether a paresis is organic or functional.

PERIODICAL PARALYSIS OF THE EXTREMITIES.

We include here a number of cases which have only recently aroused attention. Some of them, which were intermittent paraplegias, with or without anesthesia, or paralysis of the sphincters, lasting hours, and disappearing after a free perspiration, have been included under *malaria*. They occurred in quotidian or tertian types, and were influenced by quinine.

Special interest is attached to those cases of periodical paralysis of the extremities in which no relationship with malaria has been established. We include here, among others, the case described by Westphal and myself, which may be looked upon as a type of all. A thirteen-year-old *boy*, after suffering from scarlatina, had attacks of paralysis which occurred irregularly every few weeks. These *attacks* generally came on in the night, and the paralysis lasted until the next afternoon or evening. When these apyretic *attacks* were at their height, there existed a flaccid paralysis of all four extremities and of the trunk, the cranial nerves being uninvolved. The knee-jerks were absent and the electrical excitation lowered or annulled. The sensibility was normal. The patient complained of inability to move, of thirst, and of excessive perspiration. There was also some difficulty in passing the urine.

The seizures gradually lose in intensity. Between them the boy—now a man—is well, except for occasional abortive attacks and slight weakness in certain muscular regions.

In the further course of the disease I found that there existed during the attacks *a dilatation of the heart, with the symptoms of a mitral insufficiency*, which could not be found between them. The patient developed normally, married, but still has the attacks. The cause and nature of these paralytic attacks are obscure. Perhaps they are due to a virus which continually renews itself. Similar observations have been made by Hartwig, Fischl, Cousot, Goldflam, Greidenberg, Hirsh, etc. In most of these cases it was a family disease. Goldflam found qualitative alterations of electrical excitability, and claims to have discovered after the attacks toxines in the urine as well as albuminuria. Westphal and I found certain alterations in excised pieces of muscles, which we, however, did not regard as important. Goldflam found hypertrophy of the primitive fibres and vacuoles, with a glossy substance imbedded between them, and on account of these changes regarded the trouble as a muscular one. Bernhardt described it in combination with progressive muscular distrophy.

OSTEOMALACIOUS PARALYSIS.

Koppen and others have observed paresis of the muscles of the lower limbs in persons afflicted with osteomalacia, combined with pains and pain on pressure on the bones and some nerves. The gait was a waddling one, due to paresis of the flexors of the hip. More accurate data are wanting. It was thought to be due to a muscular degeneration.

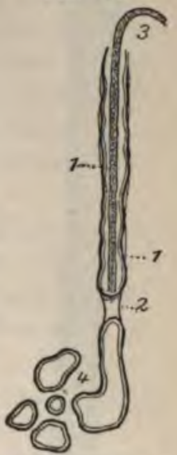
SECTION II.

DISEASES OF THE PERIPHERAL NERVES.

ANATOMY OF THE PERIPHERAL NERVES.

THE branches of the cerebrospinal nerves, as well as the trunks themselves, consist of medullated nerve-fibres with the sheath of Schwann, while only a few, or perhaps none, contain non-medullated nerve-fibres. Boveri and Kölliker believe that even the smallest have a thin medullated covering. (Fig. 154.)

FIG. 154.



Medullated nerve-fibre with Schwann's sheath (1); axis-cylinder (3); below the medullary matter has flowed out (2) and formed myelin drops (4). (After Schwalbe.)

The medullated fibres of the peripheral nerves consist of (1) an *axis-cylinder*, (2) the *medullary sheath*, and (3) the *sheath of Schwann*.

The *axis-cylinder* (neuraxon) lies in the centre, occasionally somewhat eccentric to it, and is seen only after removal of the medulla, or in colored sections through the use of reagents (for example, as a red spot with carmine, red-brown with chloride of gold, etc.). It consists of fine fibrillæ, which under a high-power microscope are shown to run longitudinally.

The homogeneous, refracting *medullary sheath* surrounds the axis-cylinder. After death it appears to have a double contour. The myelin appears as myelin drops at the nodes, and is turned black by perosmic acid.

Schwann's sheath is an elastic, structureless membrane which lies close to the medullary sheath. It contains in its upper surface oval nuclei (Fig. 155), which are elevated above the medullary sheath.

We find, in addition to the notches occurring in the fibres after death, two kinds of constrictions: (1) Ranvier's nodes, which appear at regular intervals. At these places the myelin is absent, at least for the most part, so that the sheath of Schwann extends around the whole circumference of the fibre and appears to surround the axis-cylinder directly. (Fig. 155.) The segment between two of Ranvier's nodes contains a nucleus near its middle, and it is thought that the nutritive material is absorbed at these places. (2) We also see fine notches in the myelin which seem to run obliquely to the longitudinal axis; these are called Lantermann's incisures, and are perhaps artificial.

The non-medullated fibres (gray or Remak's) are found in the sympathetic and olfactory nerves. A cross-section of a peripheral nerve (Fig. 156) shows that it is composed of numerous bundles, each of which is surrounded by a membrane consisting of concentric lamellæ (perineurium). The fibres of every bundle are held together by the endoneurium. The epineurium, which envelops the bundles and gathers them together into a nerve-trunk, is filled with fatty cells, and carries the blood-vessels, whose branches force their way through the perineurium to the inside of the bundles.

The perineurium and endoneurium contain lymphatic tracts for the nerves communicating with the central organs.

FIG. 155.

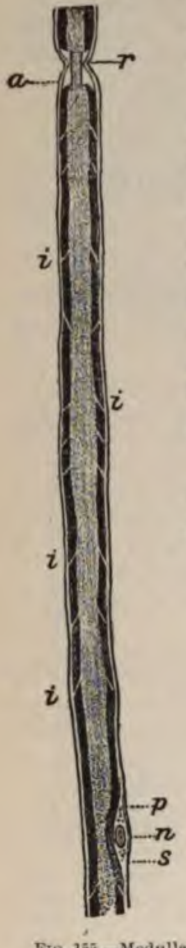


FIG. 156.

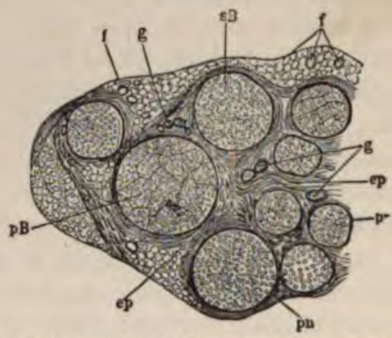


FIG. 155.—Medullated nerve-fibre. *a*, axis-cylinder; *s*, Schwann's sheath; *n*, nerve-nucleus; *p*, fine granular substance at the poles of the nucleus; *r*, Ranvier's constrictions, or nodes; *i*, *i*, Lantermann's incisures. (After Schwalbe.)

FIG. 156.—Cross-section of a nerve-trunk. pn, perineurium; ep, epineurium; sB, secondary fasciculus; pB, primary fasciculus; f, fat: g, blood-vessels. (After Seeligmüller.)

PERIPHERAL PARALYSIS.

TRAUMATIC PARALYSIS OF PERIPHERAL NERVES.

Traumata which involve a peripheral nerve produce more or less break in its continuity; and thereby disorders of motion, of sensibility, of reflex excitability, and of the vasomotor and trophic functions are set up. If slight pressure is made for a short time, perhaps for one-fourth of a minute, upon the nerve, the myelin at the place of compression may be injured, but not the axis-cylinder. The resulting disturbance of function is therefore transient. But if the compression is energetic, or if it is continued for a long time, severe alterations occur which extend through all that part of the nerve which lies towards the periphery.[1]

The alterations are more grave when a *complete section* of the nerves occurs and reunion is prevented. The cut edges retract and the *traumatic degeneration* involves the peripheral as well as the central segment. The alterations of the central segment are principally of the stump, and are characterized by a connective-tissue degeneration, with later formation of a neuroma, while the distal section is degenerated throughout its extent. After two or four days the myelin splits up into clumps and flakes, drops and balls; then the axis-cylinder disintegrates and the nuclei of Schwann's sheath become swollen and increase in number. Ranvier considers this nuclear proliferation to be the primary disturbance, and the medullary disintegration a result of it. Gradually the disintegrated material becomes absorbed, the sheaths of Schwann become filled with new nuclei and remnants of the myelin, while alterations occur in the epi- and perineurium, which, in addition to the above, produce a *sclerosis* of the peripheral part of the nerve. The intramuscular twigs and the end plates take part in this degeneration.

The degenerative changes are not, however, confined to the nerves and their branches, but also involve the *muscles*. The primitive fibres decrease in size, lose their transverse striations, their contents become granular, and at times a wax-like degeneration occurs. The nuclei of the sarcolemma and of the internal perimysium proliferate. The muscles appear pale red at first, then yellow; atrophy follows, and unless regenerative changes soon take place, they become fibrous or cirrhotic.

The less complete the break in continuity is, the easier *regeneration* occurs. Leegard noticed a re-establishment of conduction within thirty days in a case of simple constriction. Clinical observation teaches that in a paralysis caused by compression, recovery occurs in a comparatively short time after the removal of the compression (neurolysis, etc.) (Busch, Wölfler). In complete section, however, the regeneration takes a long time, except where the divided parts are not separated from each other, or where they have been sutured. The belief which previously existed, that a nerve may regain its functions by the edges of the wound healing together, without any degeneration or regeneration of the fibres, is not true according to the latest observations. (Ranvier, Vanlair, Von Büngner, and Stroebe.)

---

[1] This is not always true, as witness the results from a nerve compressed in a callus or from a bony fragment after the compression is removed. Nerve-stretching may also produce severe or transient disturbances in function (W. Mitchell).

The regeneration always commences in the central stump, from which the axis-cylinders grow into the peripheral stump (neurolization, after Vanlair). This view, although contradicted by some, explains the observations of Vanlair, Gluck, Assaky, etc., that in traumatic and operative defects of the nerves the loss of substance may be made up by the interposition of an indifferent material,—for example, a decalcified osseous tube (*suture tubulaire*), a bundle of fibres of catgut (*suture à distance*), etc. The newly-formed nerve-fibres which the central stump sends out use this intermediate substance as a bridge or support to reach the distal part. Ziegler, however, does not believe that the new axis-cylinders are formed from the old ones, but from protoplasm formed by proliferation of the nuclei of the sheath of Schwann. Clinical data do not seem to confirm the results of experiments on animals, as in some cases of nerve-suture the re-establishment of function occurred so quickly as to make it impossible that the regenerated fibres grew into the distal end of the nerve.

The fact that the restoration of function was the slower and more complete the nearer the injury was to the central ganglion seems to harmonize with the above views. Etzold observed an incomplete regeneration years after in a section of the axillary plexus; while in sections of the nerves of the arm, in the forearm, and especially the hand, the functions returned in from three to six weeks. (Langenbeck, Bruns, Tillmanns, Wölfler, Schüller, Kölliker, Schede, and others.)

The younger and stronger, the more "vital" (Raymond) the individual is, the sooner does the nerve regenerate.

The degenerative alterations of the nerves and muscles produce a loss of electrical irritability, the *reaction of degeneration*, whose course has been followed by Erb, Ziemssen, and Weiss in experiments on animals.

After a sharp preliminary increase in electrical stimulation for both currents, it decreases after the second day, to be completely lost within about ten days. Although the muscles remain unresponsive to the faradic current, an increase of galvanic stimulation is noticed in the second week, with the characteristic alterations of the reaction of degeneration.

This increase may be so considerable that, according to E. Remak, a twentieth part of the normal current strength suffices to produce contractions (0.1 milliampere on the diseased to two milliamperes on the sound side).

If regeneration does not occur, direct galvanic stimulation decreases again in from three to nine weeks, while the sluggish anodal closure contraction may be noticed a year or years afterwards with a stronger current. The slighter the lesion is, the less involved is the electrical excitability. In cases of slight compression, which may be strong enough to produce paralysis, the electrical excitability may remain normal or only decrease slightly. A slight increase may be noticed in mild affections. But then electrical stimulation *above the lesion* is lost. In some cases a partial reaction of degeneration is noticed. We also find in some cases decreased faradic with normal or increased galvanic excitation.

The symptoms produced by injuries of the peripheral nerves depend upon the position and nature of the nerve as well as upon the severity of the lesion. The most frequent injury is that of the mixed nerves. In these we have always, or almost always, *a disturbance of motility*. Those muscles which are innervated by nerves arising distally from the place of injury are completely or partially paralyzed. If they are supplied also by twigs of other nerves, paralysis may be absent. The paralysis is always a *flaccid* one; the tendon reflexes are abolished in the muscular regions involved. *Atrophy* soon follows; but, before it is noticed, degeneration may be recognized by electro-diagnostic methods.

Anesthesia does not show itself as often as the paralysis. It has been shown experimentally (Lüderitz) that slow pressure breaks the motor continuity sooner than the sensory; sensation may be intact even when there is complete abolition of motility. More remarkable is the fact that in complete section of sensory and mixed nerves the *sensation may remain intact*, or, if involved, *soon recovers*, or is confined to a more limited area than would be expected from their anatomical relations. This results from the numerous anastomoses which join together the many peripheral branches of the sensory nerves, so that many of the tegumentary zones derive their innervation from different nerves. Anastomoses of the nerve-trunks also occur, though they are few and inconstant,—for example, in the lower arm (Tessier, Gegenbaur, Létiévant, etc.). A collateral innervation has also been described,—*i.e.*, after section of a nerve, stimulation of the peripheral branch passes to a neighboring nerve through a collateral branch and ascends in it (above the lesion), to be transmitted through another branch of this intact nerve to the trunk of the injured nerve.

The fact that after section of a nerve-trunk—for example, the median—its distal end is still sensitive is referred to fibres which arise in a neighboring sensory nerve (*sensibilité recurrente*, after Arloing and Tripier, Laborde, Vanlair, Létiévant). Further, nerve-fibres may grow from the sound area into the anesthetic zone (Schuh), and sensory excitation of neighboring tactile corpuscles may also excite the sensory fibres, etc. It is thought, likewise, that the sensory fibres are more resistant and more capable of regeneration. Leegard does not believe this, and claims that for the conduction of sensory impulses a few fibres only are necessary.

H. F. Müller has especially laid stress on the fact that in peripheral neural paralysis, notwithstanding intact sensibility of the skin, a gross disturbance of electro-muscular sensibility is present. Lately attention has been called to the fact that the formation of anastomoses in the motor nerves plays a greater *rôle* than was formerly thought, inasmuch as muscular paralysis in lesions of motor nerves may be absent or soon disappear, because the muscles receive innervation from anastomoses with other nerves (Bardeleben, Frohse).

Outside of a few cases in which injuries of a mixed nerve-trunk impaired particularly the sensibility, severe, extensive, and continued anesthesia occurs, as a rule, only as a result of traumata which produce a dissolution in continuity of more than one nerve-trunk or the whole plexus.

Injuries of sensory or mixed nerves almost constantly cause *paresthesia*, and often *pains*, which are particularly marked in cases of a partial break in the continuity.

We often find a *hyperesthesia*, rarely a total anesthesia, which may include one or all the different qualities of sensation. According to Herzen and Goldscheider, compression of a mixed nerve first involves the cold and pressure senses; later, the senses of warmth and of pain.

Vasomotor and *secretory* disorders often result from peripheral nerve lesions. We find redness, with a local increase of temperature, which later may become decreased. *Cyanosis* may also follow the hyperemia. *Edema* is observed rarely; local *hyperidrosis* more often is present. We find *trophic* disorders of the skin, later of the bones and joints. The skin is occasionally smooth, shining, very thin and vulnerable, so that the slightest injury may produce ulceration. The development of small vesicles, which break and form badly-healing sores, is now and then observed. Atrophy of the subcutaneous tissue, inhibition of growth, or thinning of the bones may all occur. The nails and hair also may take part in the changes. In some cases they have been the only signs of the disease.

Motor phenomena of excitation do not play much of a part in injuries of peripheral nerves. Fibrillary tremor need not be discussed. The muscular twitchings which occur at times are probably of reflex origin. Secondary alterations, secondary contractures, atrophy of the capsule of the joint, etc., often complicate the course of the disease.

Differential Diagnosis.—*Direct traumatic muscular paralysis and muscular atrophy* following upon *contusions of the joints*,—both of which present only quantitative disorders of electrical stimulation with intact sensibility,—and especially the so-called *ischemic muscular paralysis* and *muscular contractures* (Volkmann-Leser), must be excluded. These are the muscular contractures which result from the use of excessively tight splints, especially on the upper limbs. The hand that has been left free swells and is very painful. If the splint is not removed soon, the pains increase and a flexion contracture of the hand and fingers results. A shrinking of the muscles soon follows the swelling, the muscles becoming rigid and as hard as a board. Active movements are lost, and passive ones are hindered and painful. The hardness and rigidity of the muscles, with the absence of reaction of degeneration, and the coarse sensory disturbance enable one easily to distinguish the ischemic muscular paralysis from the neuritic.

It must, however, be borne in mind that the manifold mechanical results of peripheral injuries (stiffness of the joints, scars in the muscles and tendons, etc.) may make the diagnosis of traumatic paralysis of the nerves very difficult.

Course and Prognosis.—The course depends upon the severity of the injury. A simple paralysis due to compression may recover in a few weeks, even days. If the dissolution in continuity is complete, a perfect and complete restitution cannot be expected for some months. If the stumps of the nerve are separated, a recovery can only be expected when the union is artificially aided.

Slight injuries may cause much damage in those who suffer from chronic *alcoholism*, chronic *lead intoxication*, *cachexia*, or post-infectious weakness (Oppenheim and Siemerling).

The condition of the *electrical excitability* indicates the chances for recovery. If a reaction of degeneration exists, complete recovery is doubtful, the course is a protracted one, and in the best cases recovery can only be expected in three or four months, or longer. If after the second week the electrical excitability is not involved, or only slightly, a rapid recovery may be looked for. Partial reaction of degeneration indicates a relatively good prognosis, though recovery may not be rapid. These rules, however, are not absolute. Some cases disappoint all prognostics.

In cases even of long standing therapeutic measures may do good. In one of Duchenne's cases, for example, electrical treatment proved beneficial after four years; in one of my cases, after twenty years. Secondary suture of the nerve may accomplish good results after many years.

Treatment.—The injury demands *rest* and *protection* of the limb. The injured nerve must not be irritated by pressure or stretching. The circulation must be kept free. Contraction of the muscles between which the nerve rests should be avoided. *Local antiphlogistic* treatment is indicated only when there are signs of acute neuritis.

If it is a simple compression or crush, an *electrical treatment* is the best procedure, although its value is denied by some. In recent cases I use the stabile cathode of the galvanic current on the points of pressure, the anode being placed on the plexus or any indifferent location. A current strength of six to eight milliamperes with an electrode of twenty to thirty square centimetres (Remak) should be employed, or the current strength in which the patient feels a subjective ease in movement.

If reaction of degeneration is present, combine with this a direct stimulation of the paralyzed muscles with a labile galvanic current. If the faradic excitability is not lost, the use of a faradic current may be indicated, though it is not advisable to excite the nerves directly during the first stage. Duchenne had great success with the faradic current. Even weak inductive currents which do not produce contractions are said to increase the excitability of the nerve after a certain time. It is doubtful whether excitation of the nerve above the lesion with a strong current is to be recommended.

The use of *strong currents* in recent cases should always be *avoided*, and individual sensitiveness should be taken into account. In old cases strong currents or *galvanofaradization* or *franklinization* may be employed.

Massage in the neighborhood of the compression may be combined with the electrical treatment, avoiding, of course, all mechanical irritation of the injured nerve. This is especially valuable in the later stages. Wet packs and alkaline baths are also advised.

If an *open wound* is present, the nerve must be *sutured* (free opening of the stump, freshening of the surfaces, stretching of the stump, etc.).

The statistics of Tillmanns and Schmidt—who in 1889 collected one hundred and twenty-nine cases from literature—showed that the operation produced more or less restoration of function in almost two-thirds of the cases.

Schede says that in careful suturing recovery is the rule. Recovery need not be expected before some months or a year, though it has been observed earlier in some cases. The operation is performed more often on the brachial nerves than elsewhere.

If the patient comes under treatment at a later stage, after the wound has become closed, *secondary suturing of the nerve* is contraindicated if the nerve had been cut in two. We do not have any criteria by which we may distinguish a *break* of conduction in a nerve due to a crush from a division of it. Only when the paralysis and degeneration are incomplete can we with any certainty exclude a complete break in continuity (in case the muscle is not also supplied by another nerve). An *expectant* treatment is advisable in all doubtful cases, as nerve-suturing may cause regeneration after several—even after from ten to fourteen—years. (Tillmanns.) Still, delay should not be too protracted.

Surgical works ought to be consulted for the methods of operation.

If a connective-tissue cicatrix, or a neuroma, has formed between the two stumps, it must be removed before the suturing is commenced. To compensate for the loss of substance, many suggestions have been made: union by means of indifferent material, according to the recommendations of Vanlair, Gluck, and Assaky (see above), which Schede, however, regards as only of theoretical interest; the *greffe nerveuse* (Létiévant, Phillipeau, and Vulpian), which consists in the union of the distal end of a bisected nerve with the proximal end of another of minor functional value, or, where only *one* nerve has been injured, the insertion of the peripheral end into the freshened border of an uninjured one. This is said to have been done successfully in some cases (Després, Gunn). Létiévant, however, recommends that the union should be made by nerve-flaps (*autoplastie à lambeaux*) formed by longitudinal division of the ends of the divided nerve. This operation was performed with success by Tillmanns, Kölliker, Saenger, and Sick, among others. Transplantation of nerves from animals has also been recommended.

From the above we may gather that secondary nerve-suture may be employed with good results Wölfler claims that the result is uncertain if the diastasis comprises more than four centimetres. In some cases of nerve-tumors, extirpation, followed by suture, was employed with success. (Monod, Bruns-Kredel, among others.)

It may be necessary to remove foreign bodies, especially fragments of bone, from the nerve, to free it from a cicatrix or from an exostosis or callus (neurolysis). In these cases the symptoms of a nervous lesion only appear some time after the injury. First, pains and paresthesia, then atrophy and paralysis, and finally anesthesia come on.

If conservative treatment does not cure, neurolysis is in place. Busch saw a paralysis of sixteen years' standing disappear after neurolysis. It is noteworthy that in this, as in similar cases (Schede, Wölfler, Neugebauer), improvement was noticed immediately after the operation, which indicates that the axis-cylinder was not completely diseased. Once remissions took place, due to the fact that the nerve again became bound down; for this reason it has been recommended to surround it with epidermis, fat, etc. Complete recovery requires months or even years. Röntgen's rays may be used to discover the place of a lesion when the callus is not palpable or visible. (Oppenheim.)

In cases of luxations of joints, where the nerve is involved, the joint should immediately be put in place. In one case which came under my observation, this was neglected for three days, with the result that a severe plexus paralysis developed.

Even after surgical treatment (suture, etc.), electrical treatment must be given. Do not abandon it too soon, as it may take a year until recovery is assured. Supporting apparatus can also be used to counteract the effects of the paralysis.

Finally, muscular transplantation has been advised for compensation in old paralyses.

NEURITIS—INFLAMMATION OF THE NERVES.

A neuritis may arise in the perineurium and limit itself to this part (*perineuritis*); it may have its seat principally in the interstitial tissues (*interstitial neuritis*), or it may confine itself to the nerve-fibres (*parenchymatous neuritis*). The first two forms represent a true *inflammatory process*, the last is more properly a *neural degeneration*, and produces the same alterations which we have learned to be the results of a nerve section or a secondary nerve degeneration (Waller's degeneration). A close distinction between these different forms, however, cannot be made, as they generally occur at the same time, the inflammation rarely confining itself to a single part of the nerve. For this reason, also, there is little difference in the symptomatology.

Anatomical.—An acute perineuritis causes *redness* and *swelling* of the connective tissue around the nerve. Even a spindle-shaped intumescence is noticed occasionally. The arteries in the membranes of the nerve are dilated, overfilled with blood, and may be the seat of small hemorrhages. Serous transudations and migration of white cells follow this hyperemia. The local swelling is at first caused by the exudate, later circumscribed connective-tissue proliferation produces *nodes* in the nerve at one or more places (*disseminated nodal neuritis*).

These alterations are rarely confined to the perineurium; the interstitial tissues are also infiltrated and inflamed, though perhaps only slightly; and, if it is a severe and protracted case of perineuritis, the nerve-fibres become involved.

Interstitial neuritis depends upon analogous alterations in the interstitial tissues, and may also produce local swelling, which involves almost always the nerve-fibres.

The *parenchymatous* or *degenerative* neuritis (atrophy of the peripheral nerves) is the most common form; it may be secondary to the above forms or primary.

Gombault's periaxile segmentary neuritis is a mild type of nerve degeneration in which only some segments of the nerve-fibres have broken down, the axis-cylinder being intact. Gudden believes this to be a process of restitution. It has been observed in toxic and infectious conditions.

The causes of neuritis are very numerous. The traumatic form has been already dealt with. Not only direct wounds, but contusions, stretching, and compression from forced muscular exertion may cause it. Dislocated joints, fragments of bone, bony callus, exostoses, tumors, etc., may be causative factors. Repeated though slight compression on a nerve may cause neuritis. *Crutch-palsy* and *professional neuritis* result in this way. The latter is generally caused by tools of trade, through constant use, exercising compression on the nerves of the hand and causing them to inflame. (See chapter on Occupation Pareses.)

Chemical agents, ether (subcutaneous injections), perosmic acid, etc., may cause it. The influence of cold was formerly exaggerated, but we have no right to ignore it entirely as an etiological factor. Of greater importance are the *infectious diseases*. During the course or following typhoid, variola, diphtheria, tuberculosis, syphilis (which involves the ulnar more than any other nerve), we notice a multiple, also at times a localized neuritis. *Puerperal* neuritis is generally a polyneuritis.

The chronic intoxicants (alcohol, metallic poisons, nicotine) are also factors, though they generally produce, excluding the toxic paralysis of the optic nerve, an inflammation or degeneration extending over many nerves.

Carbonic acid poisoning, ptomaine poisoning, poisoning from carbon disulphide, may also cause neuritis.

Infections and intoxications may produce such a condition of the peripheral nervous system that a slight trauma, which would not injure a normal nerve, causes a neuritis. This is due to a slight, perhaps latent, nutritive disorder, which shows itself clinically when a new noxa increases the inflammation or degeneration. (Oppenheim and Siemerling.)

Other etiological factors are *rheumatism*, *gout*, *diabetes*, *leukemia*, *arterio-sclerosis*, *arteritis obliterans* (Gombault, Joffroy, Schlesinger, and myself, among others), and diseases of the veins (Quenu). On the other hand, alterations of the blood-vessels are looked upon by some as a result of neuritis. (Lapinsky, Moltschanoff.)

Inflammation of the neighboring organs may extend to and involve the nerves as a facial neuritis in caries of the petrous bone, etc. This may occur also in inflammations of the joints. In the old *migratory* or *ascending neuritis* this was the cause of the trouble. Experimental observations have shown that an artificially-produced, purulent neuritis may progress centripetally along the ascending nerve-tracts. (Kast and Rosenbach.)

In simple, non-purulent inflammations this ascension has not been proved, though some clinical observations seem to indicate that the neuritis may ascend towards the spinal cord in the form of disseminated foci.

Inflammation produced by an injury to the fingers has been known to extend to the nerves of the arm. It, however, takes place more rarely than the older neuropathologists (especially E. Remak[1]) claimed. Lately Kausch, Krehl, and Gerhardt, among others, have described a number of such cases, and have laid stress upon the fact that they result from *infected* wounds.

Symptoms.—Acute interstitial neuritis and perineuritis may commence with *fever* and chills, though it is not the rule where only one nerve is involved. The cardinal symptom of acute neuritis is the *pain*, which is found localized or diffused, is *severe*, boring, tearing, or burning, and of long duration, though it varies in intensity. It is increased by movement, pressure, muscular action, and stretching and compression of the nerve. The skin may be reddened, but is rarely edematous. Its temperature may be elevated.

The inflamed nerve is *sensitive to pressure*, and this sensitivenes involves the whole trunk; at times mere circumscribed areas, especially where it emerges from a bony canal or a fascia or runs around a bone. The pressure does not merely produce a local pain, but the pain radiates up and down.

In many cases a swelling or a spindle-shaped intumescence of the nerve may be detected by palpation. I found, for example, in an alcoholic who complained of severe pains in the region of the perineal nerve, that this nerve was swollen to twice its normal size.

The above symptoms are those of an inflammatory process; they do not betray its nature or its physiological character, for dolor, rubor, tumor, and calor are the signs of every inflammation.

The first signs of involvement of a nerve are the *parasthesia* and *hyperesthesia* in the diseased region. Motor symptoms of irritability do not occur so constantly; these consist in fibrillary twitching, tonic muscular contractions, etc.

The tendon reflexes may be increased in recent and slight cases, but are generally decreased and *absent* entirely in a *somewhat advanced neuritis*. The electrical excitability is often increased at first, but decreases later on, as soon as there is a break in continuity.

Trophic disorders, herpes zoster, a glistening skin, and the like, are often observed. It should be remembered that decubitus, gangrene, pemphigus, and especially mal perforans (perforating ulcer), may be of neuritic origin. More rarely, swelling and inflammation of the joints and ankylosis result. Some authors consider Dupuytren's fascial contracture to be of neuritic origin.

---

[1] Many of the cases which have been referred to me as *ascending* neuritis I found to be cases of traumatic neurosis or hysteria. In only two or three did a true ascending neuritis appear to be present.

Later on, symptoms produced by an inhibition in conduction occur, —*anesthesia* or *hyperesthesia*, at times *retarded response to pain* (Erb, Kraussold, Westphal), *paralysis*, and *atrophy*. These are only absent in cases of pure perineuritis and where the process ends in recovery before deeply-invading structural alterations in the nerve parenchyma have occurred.

Chronic perineuritis, or *interstitial neuritis*, is not generally associated with increase of temperature. The phenomena develop insidiously, the pain is less severe, nor is sensitiveness to pressure so marked. Otherwise what has been said for acute neuritis holds true.

Parenchymatous or degenerative neuritis presents less evidence of its true nature than any other type. Here we have, above everything else, signs of a break in continuity. *Hyperesthesia* and *flaccid degenerative paralysis* in a definite nerve-zone are the characteristic symptoms, though *pains* and *paresthesia* are also generally present. The nerve is sensitive, but not as much so as in the other forms. There is also no swelling of the nerve. The mingling of all these types has already been spoken of. Neuritis may become a systematic disease,—*i.e.*, confine itself to the sensory or to the motor fibres of a nerve.

Course and Progress.—Acute neuritis may be recovered from in a few weeks, but it more often lasts longer, or a chronic neuritis develops. The primary chronic cases are lingering and protracted ones, and may last for years. The *prognosis* is better in acute cases, especially in those of rheumatic or toxico-infectious origin. In general, it depends upon the severity of the process, and in secondary neuritis upon the character of the original trouble.

The tendency of neuritis to extend, even as far as the spinal cord, has been overvalued.

Treatment.—In recent acute cases, besides the proper and necessary treatment of the wound itself, *rest* is the first requisite, as every movement and every muscular contraction may increase the inflammation. The diseased member should be immobilized in such a manner as to guard against all pressure and all stretching of the nerves.

A *general diaphoresis* is especially advisable in rheumatic and infectious cases. Local applications of *cold* (ice or cold-water compresses), *leeches*, and inunctions of *gray ointment* can be used. If the irritation is less intense, *wet packs*, *hot compresses*, or hot sand-bags may be employed. These are likewise beneficial in subacute and chronic cases; vesicants can also be used in such cases, but they should not be applied to anesthetic zones. The diet should be light and non-irritating Laxatives must be given if necessary.

The drugs that are used are sodium salicylate, antipyrin, mercury, and potassium iodide (in syphilitic cases especially). Subcutaneous injections of morphine may be necessary to subdue the pain.

Faradic currents should *never* be used in the acute stage. A careful galvanic treatment may, however, be of benefit, especially the stabile use of the anode upon the chief points of pain, the cathode resting upon a central part of the nerve. (Current strength to be two to four milliamperes with an electrode of about twenty square centimetres.) Some recommend stronger currents, but they should be used only in subacute and chronic cases. They should, however, be avoided in the stage of irritability.

Massage and electricity may accomplish wonders whenever the signs of irritation have disappeared, or where they have been absent from the beginning or if the case is a lingering one. We can use, in addition to stabile applications of the cathode to the painful parts, labile galvanic muscular stimulation, and even the faradic current, provided faradic irritability is not abolished. Massage should never be used in the first stage. The atrophy, contractures, and shrinking of the fasciæ should be combated by massage and *active* and *passive movements*.

An *operative* treatment (nerve-stretching, division, etc.) is sometimes indicated in old cases of interstitial neuritis or perineuritis. Recovery in chronic cases may be hastened by the use of *thermal baths*. (Wiesbaden, Teplitz, Wildbad.)

PERIPHERAL PARALYSIS OF SPINAL NERVES.

PARALYSIS OF THE PHRENIC NERVE.

This nerve is not often paralyzed. Excluding diseases of the cervical cord which involve its nucleus, this nerve is paralyzed most often by injuries which affect the third and fourth *cervical roots*. These are spondylitis, fractures and luxations, vertebral tumors, pachymeningitis, spinal hemorrhage, and especially syphilitic meningeal diseases. The nerve is rarely involved in injuries of the neck, on account of its protected position. The same is true in *tumors* of the neck or thorax.

Neuritic paralysis of the *phrenic* may be of *rheumatic* (?), *toxic*, or *infectious* origin. A bilateral phrenic paralysis may occur in the course of an alcoholic neuritis, also from lead intoxication (Duchenne), and as a post-diphtheritic paralysis.

Myositic paralysis of the diaphragm, which is generally due to an extension of an inflammation of the pleura or peritoneum, need not be discussed here. Whether the paralysis of the diaphragm observed by Gerhardt in a case of tabes was of central or peripheral origin is not known.

The symptoms of paralysis of the phrenic are those of *paralysis* of the *diaphragm*. The diaphragm does not contract during inspiration, in consequence of which the epigastric arch is absent; the sinking of the diaphragm in inspiration is not detected by palpation, and the lower border of the liver is retracted. In expiration the hypochondriac regions and stomach protrude and the liver is depressed. The diaphragm may be pressed upward, also the lower border of the lungs. If the paralysis is incomplete, a slight pressure is sufficient to press back the descending diaphragm in inspiration.

Dyspnea occurs on slight exertion. This may become very pronounced. It is dangerous when bronchitis or pneumonia come on, and increased demands are made on the respiration. Not only inspiration but also expiration suffer, as the air breathed in is not sufficient, and the abdominal muscles do not act energetically enough when from the flaccidity of the diaphragm the abdominal cavity becomes enlarged. *Unilateral* paralysis of the phrenic is not easily recognized, as the disturbance of function is slight. With careful examination it may, however, be discovered. The absence of the so-called *diaphragmatic phenomenon* (Litten)—*i.e.*, the objective movement of the diaphragm in inspiration and expiration—may be of diagnostic interest. The results of *electrical exploration* are also of moment. The respiratory sounds are heard but slightly at the base of the lung (Suckling). The electrical excitability may be lost for both galvanic and faradic currents to be restored in convalescence. In neuritis of the phrenic we may find a painful pressure-spot on the scaleni, between the two bellies of the sternocleidomastoid, or external to them.

It is not certain that there is a true *hysterical paralysis* of the *diaphragm*. It is imitated by hysterical patients, due to the fact that they use the thoracic muscles, especially the upper costal, excessively, at the same time not calling upon the diaphragm for action. By careful observation a protrusion of the epigastrium is occasionally noticed, thus showing the character of the disease. *Dyspnea* is also absent, and the mind has a great influence upon the disorder. Wernicke speaks of an hysterical insufficiency of the phrenic which is combined with fear and inspiratory dyspnea.

The prognosis is good in all cases except where the phrenic becomes involved in multiple neuritis.

Treatment.—Treat the cause by removal of tumors and by electrotherapy. In neuritis apply counter-irritation over the nerve (Suckling).

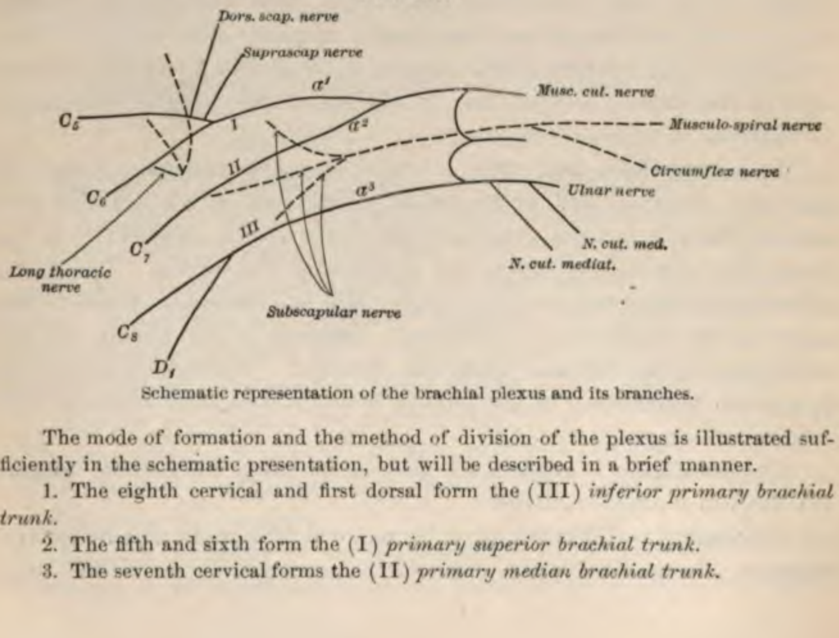
In diphtheritic paralysis of the diaphragm strychnine injections are recommended. Employ general treatment for multiple neuritis, etc.

PARALYSIS OF THE BRACHIAL PLEXUS. COMBINED PERIPHERAL PARALYSIS OF THE NERVES OF THE SHOULDER AND ARMS.

On account of the difficulty in comprehending the anatomical relations of these nerves and their practical importance, I consider it advisable to give the anatomy, and shall follow the description which Schwalbe gives in his text-book of anatomy.

The anterior branches of the *fifth* to the *eighth cervical nerves* unite with the *first dorsal* to form the brachial plexus. Its roots, which increase in size downward, are placed between the origins of the scalenus anticus and scalenus medius, at their point of exit from the intervertebral foramina. The three upper roots descend from here, the eighth cervical runs horizontally, and the nerves from the first dorsal take at first an ascending direction. They all soon unite at an acute angle and form the plexus. It lies between the anterior and middle scaleni, with its three upper roots above the subclavian artery and the two lower ones behind it. From here it descends obliquely through the supraclavicular fossa, passes under the clavicle and subclavian muscle, covered by the major and minor pectorals, and reaches the arm-pit. Here it lies between the subscapular and serratus anticus, and terminates in its long brachial branches. The axillary artery lies below the clavicle, in front of the median portion of the plexus, and in the axillary space passes through the opening between the two roots of the median nerve to reach the posterior side of these nerves. Many *variations* occur in regard to the formation of the plexus and its further course; but a general type of ramification is observed, which is presented in Fig. 157.

FIG. 157.



Schematic representation of the brachial plexus and its branches.

The mode of formation and the method of division of the plexus is illustrated sufficiently in the schematic presentation, but will be described in a brief manner.

1. The eighth cervical and first dorsal form the (III) *inferior primary brachial trunk.*
2. The fifth and sixth form the (I) *primary superior brachial trunk.*
3. The seventh cervical forms the (II) *primary median brachial trunk.*

Each of these primary trunks divides into an anterior and posterior branch, which unite and form three new cords.

1. A superior lateral one, the outer cord, which gives off the musculocutaneous and the outer head of the median.
2. An inferior median one, the inner cord, which gives off the cutaneí nerves, the ulnar and the inner head of the median.
3. A posterior one, the posterior cord, which gives off the circumflex and musculospiral nerves.

The short nerves of the plexus which pass to the shoulder are the *posterior thoracic* nerves, the dorsalis scapulæ (to the major and minor rhomboid muscles), the long thoracic (to the serratus anticus), the subscapulæ (to the subscapularis, teres major, and latissimus dorsi), and the circumflex nerve. The suprascapular are also short nerves and enter the supraspinatus and infraspinatus muscles.

The anterior thoracic nerves supply the subclavius and the pectoralis major and minor.

Isolated diseases of these nerves may occur, as well as *plexus paralysis*. A close distinction between root and plexus involvement cannot be made, as we cannot always decide whether the roots are involved before their union to form the plexus or afterwards. (Pagenstecher, Raymond.)

Injuries of the shoulder, or a push or a blow directed against the supraclavicular fossa, punctured and sword wounds, a fall on the shoulder, etc., all may injure the whole plexus or single parts of the roots. This is true especially in *luxations* of the *humerus*, *fractures* of the *joint* and of the *clavicle*, etc.

Tumors of the upper clavicular region may compress the plexus and produce paralysis, as I noticed in a case of aneurism and arteriosclerotic dilatation of the subclavian artery.

A primary neuritis of the brachial plexus (rheumatic, toxic, infectious) also occurs.

Obstetrical paralysis and *shoulder dislocations* produce a compression paralysis, which will be separately described.

The nerves of the arm, after their exit from the plexus, are almost always singly attacked; but may be injured together through constriction of the arm (gymnasium rings, Esmarch's bandage, handcuffs, gum bands, etc.).

The most important *partial paralysis* of the *plexus* is

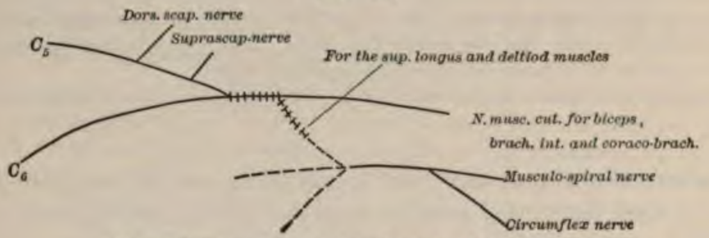
(DUCHENNE-) ERB'S COMBINED PARALYSIS OF THE SHOULDER AND ARM.

It always involves the *deltoid*, *biceps*, *brachialis internus*, and *supinator longus*, often the *supinator brevis*, occasionally the *infraspinatus*, and rarely the *subscapular*.

This paralysis results from lesions of the fifth and sixth cervical roots or the superficial parts of the primary plexus trunks which result from the union of the fifth and sixth cervical roots. (See scheme, Fig. 158.)

This paralysis is produced by *traumata* which directly affect the plexus, or which by forced adduction of the shoulder on the vertebral column press the clavicle against the plexus. This is particularly the case in forcible movements of the elevated arm outward and backward.

FIG. 158.


Dors. scap. nerve
Suprascap-nerve
C_5
For the sup. longus and deltiod muscles
N. musc. cut. for biceps, brach. int. and coraco-brach.
C_6
Musculo-spiral nerve
Circumflex nerve

Schematic representation of the location of Erb's paralysis.

Formerly it was believed (Hoedemaker, Nonne, Schultze) that the fifth and sixth cervical nerves were thereby compressed between the clavicle and the spinal column. Now (Wigand, Büdinger, Kron, Gaupp) it is assumed that the compression occurs, for the most part, between the clavicle and the first rib. The lesion occurs more easily if the head is turned at the same time towards the opposite side.

Many obstetrical paralyses occur in this way. Also in the so-called narcosis paralysis (Braun) it is a factor, as during a long-drawn-out operation (laparotomy) the arms are held backward and upward.

Bernhardt observed a bilateral Erb's paralysis occur in this way. Braun called attention to the fact that narcosis paralysis can also be produced by the head of the humerus pressing on the nerves in the axilla. Some are inclined to include here all forms of paralysis occurring in narcosis, whether of cerebral or spinal origin (central) or due to chloroform intoxication, etc. Hemiplegias during narcosis, due to cerebral hemorrhage and probably also to anemic softening, have even been included under this name by some (Hofmokl, v. Rüdinger).

Pressure on the above-named nerves by the carrying of burdens on the shoulder—stone-carriers (Rieger), hod-carriers (Osann), etc.—can produce an Erb's paralysis. Finally, a primary toxic or infectious neuritis may limit itself to the fifth and sixth cervical nerves, as I have seen it do more than once.

Krafft-Ebing observed a bilateral paralysis of this nature. Heyse observed it once in a consumptive who was a stone-carrier. L. Mann observed Erb's paralysis occur from ptomaine poisoning.

The disorders in function produced by paralysis of the fifth and sixth cervical nerves have already been described. We repeat the main facts here: The arm cannot be adducted (paralysis of the deltoid); it may be at times raised slightly forward, provided the small nerve-twigs which the anterior thoracic supply to the anterior portion of the deltoid are intact. The arm is extended; it cannot be flexed at the elbow-joint (biceps, internal brachial, supinator longus). If the supinator brevis is involved, the lower arm and the hand are in a position of pronation, and the hand cannot be satisfactorily supinated.

If the infraspinatus is paralyzed, the arm is turned inward and cannot be properly rotated outward.

The paralysis is almost always an atrophic one, with a complete or partial reaction of degeneration. Pains are sometimes present.

The *sensibility* was intact in many cases, even in the region of the axillary nerve, or the anesthesia had disappeared at the time of the examination. In others a disorder of sensation was found in the zones innervated by the *circumflex* and *musculocutaneous* nerves,—*i.e.*, on the *outer surface of the upper arm*, above the *median portion* of the *deltoid*, not quite to the acromion, and on the *outer surface of the forearm*. At times the sensory fibres of the median for the thumbs and index-finger were involved. There are cases of upper plexus paralysis which deviate from Erb's type. Rose, for example, in extirpation of a neuroma, was compelled to resect a large extent of the fifth and sixth cervical roots, and noticed thereupon an involvement of the scalenus, subscapularis, teres minor and major in the Erb's paralysis which resulted. Heyse found an involvement of the long thoracic nerve.

The prognosis depends upon the severity of the lesion. Recovery does not always occur, and the course is generally slow. As prophylactic procedures, the arms should not be raised during operations, nor should the head be turned to the side.

Concerning **treatment** see page 246.

Less common is the

LOWER PLEXUS PARALYSIS (KLUMPKE),

which is due to disease of the eighth cervical and first dorsal roots. Cases of this kind have been seen by Flaubert, Seeligmüller, Klumpke, Pfeiffer, and myself, among others. It may be produced by tumors, an osseous thickening of the first rib (Müller), operations, syphilitic meningitis (Dejerine), or primary neuritis of these roots. (Feinberg, myself, etc.) It may also accompany a total plexus paralysis; it may be the *residuum* of a former total plexus paralysis.

The *small muscles* of the *hand* and a part of the muscles of the forearm, especially the *flexors*, are found paralyzed. *Sensory disturbance* is generally present in the region of the *ulnar nerve*, as well as the *inner surface of the upper arm and forearm.*

The anesthesia of the hand may extend to the area innervated by the median. In one case it did not extend above the elbow-joint. (Müller.) Vasomotor disorders often develop. *Oculopupillary* symptoms only develop when the roots are injured before they leave the rami communicantes. It should, however, be observed that traumata of the shoulder or arms may, through *laceration*, produce a lesion of the root near the spinal cord. Cases are on record where forcible attempts to reduce a dislocation of the shoulder-joint tore away the roots from the cord.

TOTAL PLEXUS PARALYSIS

is in general a rare disease, and almost always of *traumatic*, rarely of *neuritic*, origin. It may be caused in delivery of a child, by fracture of the humerus or of the clavicle, by subcoracoid or axillary luxation of the shoulder-joint, by forcible attempts to replace dislocations, and by hemorrhage.

The paralysis may embrace all the nerves or be limited to some of them. Or an originally total paralysis may gradually limit itself to a definite area, often to the *axillary* and *radial* regions. This paralysis is always a degenerative one. Disorders of sensation are always present, though varying in extent and degree. The fact that the inner surface of the upper arm at times does not show any loss of feeling is due to its vicarious innervation by the intercostohumeral nerve.

The prognosis is not very good, as some paralysis and atrophy not rarely remain, no matter how far regeneration extends. This regeneration may go on slowly for years.

In a patient who had luxation of the humerus which was overlooked, and only replaced after three days, atrophy and paralysis of the whole arm developed, which partly disappeared in the first few weeks. The paralysis of part of the ulnar and of the musculospiral continued the longest, improvement still going on two years afterwards. Alcoholism was a complication.

Plexus paralyses resulting from *fracture of the clavicle* are either a direct result of force, or of the pressure exerted by the fragments, or of hemorrhage; or they may not occur until callus forms. Generally the whole plexus is involved, though the ulnar remains free in a number of cases. (Chipault.) Generally the pectoralis major is also paralyzed.

The treatment should be surgical, if deemed necessary: removal of the splinters, resection of the callus, suturing of the bone (Chipault), etc.

Paralysis resulting from the *use of Esmarch's bandage* (Langenbeck, Frey, Braun, Bernhardt, Neugebauer) involves all nerves of the arm, or perhaps only single ones. It occurs especially in emaciated persons, as well as in those of weakened constitution (from toxins). As a rule, the paralysis is slight or only moderately severe. In two cases observed by me complete recovery resulted.

OBSTETRICAL PARALYSIS.[1]

Excluding the facial paralysis caused occasionally by the forceps, we find that it is particularly a paralysis of the brachial nerves which occurs intra partum. Generally the deliveries were difficult ones in which instruments were used. In head presentations the finger or tenaculum introduced into the axilla to facilitate delivery may exercise direct pressure upon the nerves, or force the shoulder and clavicle upward and backward, thus compressing the nerves. Or the forceps may cause it, or pressure on the shoulders to hasten the birth of the head. It may also be caused in freeing the elevated arm, when a tenaculum or finger is inserted to force the arm downward or to exercise traction upon it.

The Prague or Smellie's method has also been blamed. In some cases the umbilical cord, when wrapped around the neck, has been said to have compressed the plexus.

In normal labors an obstetrical paralysis rarely occurs, and only when the child is very large and broad, thus impeding the passing of the shoulders. An imperfect pelvis naturally conduces to this condition.

The typical obstetrical paralysis was described by Duchenne. The deltoid, biceps, brachialis internus, supinator longus, supinator brevis, and infraspinatus are affected. The humerus is rotated inward, the forearm is extended, and the hand pronated. Atrophy soon follows. There are no sensory disorders, as a rule.

In breech presentations this form of paralysis almost always occurs (Peters), while in head presentations the whole plexus is involved at times. In some cases, especially where severe injuries occurred, a total paralysis of the plexus or its roots can occur. (Seeligmüller, Jolly, myself, and others.) Sensory disorders are also present in these cases; only the median region of the axilla or the whole inner surface of the upper arm are, as a rule, spared.

---

[1] The paralyses produced in labor and affecting the mother are not considered here. Some have classified infantile obstetrical paralyses into cerebral, spinal, and peripheral (Köster). The first two are mostly the results of hemorrhage (Lietzmann, Ruge, Mauthner). A rare form of peripheral obstetrical paralysis is that of the levator palpebræ supinator and rectus supinator produced by the forceps (Nadaud, Berger).

In a case observed by Seeligmüller and one seen by me the *oculo-pupillary* fibres were involved. Paralysis of the suprascapular nerve seems to be the most constant of all.

Honne and I proved anatomically that in a case of typical obstetrical paralysis the lesion involved Erb's point. I found a degeneration of the fifth and sixth cervical roots which extended along the circumflex, musculocutaneous, and musculospiral nerves.

The obstetrical paralysis is rarely bilateral. Jolly has described an uncommon case of this kind; in it the muscles of the upper arm supplied by the fifth and sixth cervical were sound, while all the muscles of the forearm and the hand, as well as the pectoralis major, latissimus dorsi, and triceps, were involved. The contraction of the antagonists (deltoid and biceps) produced a peculiar position of the arms. Jolly believed it to have been produced by a lesion of the roots of the cord, especially the seventh pair, and that the lordosis of the cervical column resulting from the face presentation produced a laceration of the roots.

The complications of obstetrical paralysis are fractures of the humerus and clavicle, dislocations of the shoulder, hematoma of the sternocleidomastoid, and chipping of the epiphyses. Küstner believes that the latter often simulates an obstetrical palsy, particularly paralysis of the infraspinatus. This condition, however, produces local pain and slight crepitation.

The prognosis is not a bad one. The paralysis may disappear in a few weeks, or may last for months, and then subside somewhat. I saw complete recovery in three cases. Seeligmüller, Bernhardt, d'Astros, Jolly, and others observed only partial restitution. Some cases may never recover. If a reaction of degeneration exists, the prognosis is worse. It is, however, hardly possible to recognize this alteration of the electrical excitability in the first five weeks. At any rate, it is advisable to commence electrical treatment as early as possible.

PARALYSIS OF INDIVIDUAL NERVES OF THE SHOULDER AND ARM.

THE LONG THORACIC NERVE

may be injured by a trauma in the suprascapular region or in its axillary course. It can occur from injuries to the shoulder, blows or falls on the shoulder, an axillary wound, from the carrying of heavy burdens, and *over-exertion*, especially work that demands continual elevation of the arms.

Men are more often attacked than women, and the right side more than the left.

It has been observed to follow *diphtheria* and *typhoid* (Nothnagel, Bäumler), and influenza (Bernhardt). It is said to follow rheumatism. I saw it occur in the *puerperium*. It does not, as a rule, take part in the upper plexus paralysis. Verhoogen's idea of an hysteric paralysis of the serratus seems to me to be improbable.

The symptoms are those of paralysis of the *serratus*. (See pages 26 and 27, Figs. 5 to 9.) Disorders of sensation are not, as a rule, present; pains may occur. We find the reaction of degeneration in severe cases.

The prognosis depends upon the cause. It is good in all but the severe traumatic cases. Recovery may be delayed for months.

PARALYSIS OF THE CIRCUMFLEX NERVE.

Fall or injury, even a fall upon the hand with contusion of the shoulder, compression of the nerve, as from crutches, rheumatism, and infection, are the commonest causes. There is a form of lead paralysis limited to the deltoid. In a case of chronic saturnism I saw a paralysis of the circumflex with sensory disturbance set in after the patient had for some time carried burdens on his shoulders. Diabetic neuritis also may involve the circumflex nerve alone.

Raymond described a bilateral paralysis of this nerve which resulted from continued elevation of the arms (in sleep). The paralysis resulting from dislocations rarely involves the circumflex alone. This circumflex paralysis is either purely motor—there existing a paralysis of the deltoid, and perhaps of the teres minor—or anesthesia may also be present.

In its further course a relaxation or an ankylosis of the shoulder-joint develops. Whether trophic disorders in the region of the nerves of the joint are a factor is doubtful. A primary (rheumatic, arthritic) ankylosis of the shoulder should not be confused with deltoid paralysis. In the first the arm is rigid at the shoulder-joint and the scapula follows the movements of the arm. Further, the patient can contract the muscles, though no movement results, and there are no degenerative signs or disorders of sensation. In muscular paralysis (with atrophy) caused by direct blows upon the muscle there is no reaction of degeneration or disorder of sensation.

A paralysis confining itself to the musculocutaneous nerve has only been observed in a few cases (Erb, Bernhardt, Windscheid, Strauss), once after extirpation of a tumor of the supraclavicular fossa, in another case after luxation of the humerus, and once from pressure caused by the sharp corner of a marble slab carried on the shoulder. I observed it once after a sabre-wound of the axilla, and in another case in fracture of the head of the humerus.

The symptoms were: *paralysis of the flexors of the forearm*, with the exception of the supinator longus, and *hyperesthesia* in the region of the *lateral cutaneous nerve* upon the *external surface of the forearm*.

The coracobrachialis was not involved in several cases. The brachialis internus, which is supplied by branches of the musculospiral, need not be paralyzed, so that the paresis in one case was limited to the biceps.

If the paralysis involves, as it generally does, all the flexors with the exception of the supinator longus muscle, the forearm can be flexed only by means of this muscle or with the assistance of the flexors of the hand and fingers. In a supinated position, flexion is impossible or considerably impaired. Electrical stimulation of Erb's point produces only a contraction of the deltoid and the supinator longus. The muscular atrophy produced causes a characteristic depression upon the outer surface of the upper arm, between the insertion of the deltoid muscle and the origin of the supinator longus.

In one of my patients the zone of hyperesthesia extended almost to the lateral border of the forearm. There also appeared to be trophic disturbance of the skin, which extended beyond the sphere of innervation of this nerve.

The course depends upon the severity of the lesion. In one case in which a moderate degree of reaction of degeneration had been detected, recovery occurred after three months' galvanic treatment.

More rare still is an isolated paralysis of the suprascapular, which I observed once. Cases have been recorded by Bernhardt, Hoffmann, Sperling, Beuzler, and Köster. It was supposed to have been caused by cold, a fall upon the shoulder, a fall upon the hand with contusion of the shoulder, and in my case by the carrying of heavy burdens upon the shoulder. It occurs more often in combination with paralysis of the circumflex and in Erb's paralysis.

The symptoms are those of paralysis of the supra- and infraspinatus with atrophy. Atrophy of the infraspinatus reveals itself very distinctly. In my patient the spine of the scapula showed very distinctly, on account of the muscular atrophy. (Fig. 159.) Electrical examination will also easily reveal the loss of action of the infraspinatus.

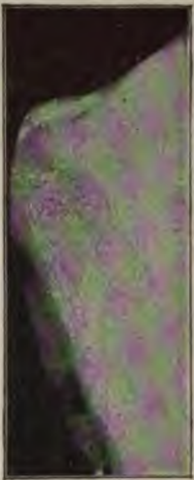
According to Duchenne, the supraspinatus fixes the head of the humerus in its socket when the deltoid is in action. It also helps the deltoid in raising the arm from the side, its action being upward and forward. When paralyzed we have some difficulty in executing this movement, and especially early fatigue (and pain) in lifting the arm, carrying burdens, etc.

Paralysis of the infraspinatus hinders outward rotation, though, according to Bernhardt, the teres minor can partly assume its functions. In my case this movement of the infraspinatus was only weakened.

Duchenne says that writing, sewing, and similar movements requiring the rolling outward of the arm, are particularly impaired.

Bernhardt has described a paralysis, confined to the plexus fibres for the supinator longus, caused by a knife-wound of the supraspinous fossa. In a case of musculospiral paralysis after a cutting injury, I observed all the muscles recover except the supinator longus, this isolated paralysis being therefore the residuum of a total paralysis of the musculospiral.

FIG. 159.



Atrophy of the supraspinatus and infraspinatus as a result of paralysis of the suprascapular nerve.

PARALYSIS OF THE MUSCULOSPIRAL NERVE.

The musculospiral nerve is paralyzed more frequently than any other nerve of the extremities. This is, in the first place, due to its peculiar course and its superficial position. It is particularly exposed to external force where it winds around the upper arm, between the triceps and humerus, and between the brachialis internus and supinator longus, especially as it is not protected by muscular tissue in this region, and lies upon a bony surface which does not permit any giving way to pressure. It is, then, not remarkable that Remak in two hundred and forty-two cases of peripheral nerve paralysis of the upper extremity, found the musculospiral involved one hundred and five times.

We do not propose to treat here of its involvement in plexus paralysis (see previous chapter), but of paralysis of its trunk and branches after the nerve leaves the plexus.

In the majority of cases it is of *traumatic* origin, and the traumata, as a rule, involve the nerve as it winds around the upper arm. Even simple pressure at this place suffices to impair its function. *Pressure paralysis* can generally be referred to the fact that the nerve *during sleep* is compressed by the head resting upon the arm, or by an arm being supported upon a hard surface. For this reason it occurs more rarely in bed than in sleeping upon the ground, upon a board, etc. Another causal factor comes generally in play. Those who are stricken with this form of paralysis are generally individuals whose peripheral nerves are already slightly impaired, particularly *alcoholics;* and it is not only the *deep* sleep into which they fall which causes it, but also the already existing, though slight, degeneration of the nerves. Chronic *lead intoxication* is also a predisposing cause. Convalescence from fevers, cachexia, and *senility* are also predisposing factors in the production of traumatic peripheral paralysis of the musculospiral nerve. In many cases it is a *toxico-traumatic* paralysis. In some the hand in grasping the arm during sleep, or in the carrying of a burden, caused the paralysis.

It may also occur in narcosis, not only in the manner described by Braun, but also from the bone being pressed against a hard support during deep narcosis.

Handcuffing, pressure bandages, heavy burdens supported on the arms, iron pitchers whose handles are carried by the upper arm, suspension, etc., can cause a unilateral or bilateral paralysis of the musculospiral. Severe *muscular action,* particularly *sudden extension* of the arm, as in throwing a base-ball, may completely paralyze this nerve. I treated a man who, in danger of falling from a ladder, suddenly and forcibly extended his arm to save himself, without, however, accomplishing his purpose. The contraction of the triceps, and, perhaps, also laceration of the nerve, resulted in musculospiral paralysis. Similar observations were made by Gowers, and Gerulanos especially has lately shown that sudden forcible contraction of the triceps muscle, particularly of its external head, which lies directly upon the musculospiral nerve, may produce a paralysis of it.

Bernhardt reports that this paralysis can also result from falling upon the back of the hand. It is needless to mention that the nerve can be injured by stabs, blows, bullets, etc. It is often injured in *fractures of the humerus,* as well as of the bones of the forearm, from bone-fragments, splinters, and callus. In one of my cases it was kept stretched by a splinter like a string on a violin bridge. It is rarely paralyzed by itself in luxation paralyses, and occasionally in connection with the circumflex nerve; *vice versa,* these nerves may be the only ones spared. In *crutch-palsy,* also, it is rarely paralyzed alone; in such cases it is a total paralysis, involving, likewise, the triceps. Crutches which produce such severe pressure are generally poorly made and not padded.

Other etiologic factors are of little moment as compared with traumatic influences. *Over-exertion* of the muscles innervated by the musculospiral nerve, with an existing predisposition, may evoke such a paralysis, as I, for example, observed in a weaver who in his work was obliged continually to extend his left hand. (See chapter on Occupation Pareses.)

Rheumatism can but rarely evoke a musculospiral paralysis. *Infectious agents,* producing a neuritis or a degenerative atrophy of the nerves, is a more potent factor. Paralysis of the musculospiral nerve has been observed in the course of *typhoid fever* (Bernhardt), the *puerperium,* and after *articular rheumatism* (Kast); here also the infectious disease appears to have made the nerves fragile, so that slight traumata may cause the paralysis. I saw it occur in a drinker in the course of typhoid fever. In the beginning of tabes dorsalis a temporary musculospiral paralysis was observed in a few cases.

The *toxic* forms of neuritis rarely, with the exception of the saturnine, confine themselves to the musculospiral nerve. These types are discussed elsewhere. In chronic arsenical poisoning and argyria (Gowers) the neuritis can likewise limit itself to the muscles innervated by the musculospiral nerve. According to Michaut it occurs also in opium-smokers.

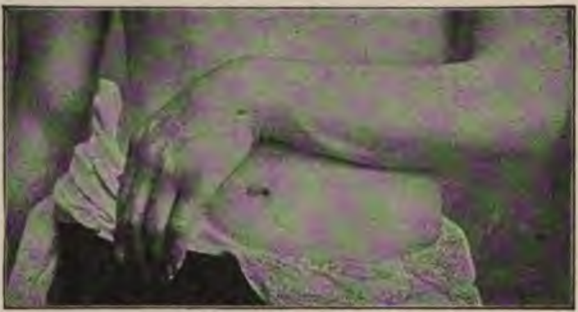
The paralysis of the extensors, often observed after subcutaneous injections of ether (more rarely of chloroform, alcohol, antipyrin, and osmic acid), is due to a direct *chemical alteration* of the nerves. It is not due to injury of the nerves by the hypodermic needle, but the ether produces inflammatory and degenerative alterations of the nerves.

Kausch described a case of musculospiral paralysis observed by him as one of *ascending neuritis.* I observed a case in which, after an injury of the thumb, a musculospiral paralysis developed. The patient had treated the wound with applications of urine. Suppuration did not, however, occur, and in addition influenza had preceded the paralysis.

The symptoms of musculospiral paralysis are somewhat dependent upon the location of the lesion. In ordinary pressure paralysis in which the nerve is involved, after the giving off of the triceps branches, the following muscles are affected: the *supinators,* the *extensors of the hand,* the *extensor communis digitorum,* together with the *indicator* and *extensor minimi digiti,* the *extensors* and the *long abductor* of the *thumb.* The triceps and anconeus are therefore the only muscles spared. The position of the hand may immediately reveal the paralytic condition (Fig. 160). The hand is flexed at the wrist-joint almost *ad maximum,* and when brought out of this position falls back into the same position when released. The fingers are flexed in the metacarpo-phalangeal joints; the thumb is opposed to the fingers and is somewhat depressed downward.

The hand cannot be extended nor can the basal phalanx of the fingers. Extension of the other phalanges is not restricted, as they are under the control of the non-paralyzed interossei. This is seen most clearly if the basal phalanges be brought passively into a position of extension. Abduction and adduction of the fingers is still possible, but on account of the flexed position of the fingers is only seen distinctly when the hand and fingers rest upon some support. It is also necessary to bring the hand to a position of extension to show the lateral movements of the wrist-joint, as it is only in this position that even a normal person is able satisfactorily to execute this movement.

FIG. 160.



Position of the hand in musculospiral paralysis.

The hand and fingers can be flexed in a normal manner. The grip is, however, considerably weakened. This is due to the abnormal position of the hand and fingers, as the flexors can only contract energetically when as a result of hyperextension of the hand their points of insertion are as far as possible from one another. The grip in musculospiral paralysis becomes normal when the hand is passively held hyperextended. Abduction of the thumb is lost or is slight, also hyperextension.

Paralysis of the supinator longus causes some impairment of flexion of the forearm. The brachialis internus, which receives some branches from the musculospiral, is, as a rule, not appreciably involved. The supinator longus paralysis can be easily detected when, on placing the forearm midway between pronation and supination, forcible—against resistance—flexion does not bring the muscle-belly of the supinator into view. Paralysis of the supinator brevis causes inability to supinate the hand with extended forearm; it is, therefore, generally in a position of pronation, and supination is only possible with flexed forearm by means of the biceps, or is replaced to a certain degree by outward rotation of the upper arm (infraspinatus).

The triceps is involved in crutch-palsy, sometimes also in luxation paralysis. An *isolated paralysis of the triceps* was observed by Seeligmüller in a case of fractured humerus, and one by myself from overexertion of this muscle.

If the lesion is in the forearm, the supinators and also the extensors carpi may be spared. In *ether paralysis*, for instance, only the extensor communis digitorum or some of its branches and the abductor longus pollicis, and not rarely some sensory branches of the skin, are involved. It is only a rare occurrence for the nerves to be compressed below the giving off of the branches for the supinator. In a paralysis from dislocation the supinator longus may be spared. In lead paralysis the supinators are, as a rule, spared, sometimes also the abductor longus pollicis.

In the common pressure paralyses we have a simple paralysis,—*i.e.*, alterations in electrical excitability are almost always absent; it is only in a few cases that we find a simple decrease or a slight increase in excitability. Electrical stimulation above the seat of the lesion, even with normal excitability, is without any effect (Erb). For instance, on stimulating the musculospiral in the axilla—at the upper end of the coracobrachialis—only the triceps contracts; upon stimulation of Erb's point, the supinators fail to contract.

In severe lesions of the nerves (wounds, lacerations, crushing by dislocated bones, etc.) a reaction of degeneration develops. If the crushing is only moderately severe—crutch-palsy, slight degrees of luxation paralysis, in some cases also in pressure paralysis—a partial reaction of degeneration may generally be expected. The muscular condition corresponds to these disturbances: the muscles retain their normal volume in cases of slight paralysis; in severe cases more or less *emaciation* takes place, which is particularly prominent upon the extensor side of the forearm. We occasionally observe a *swelling* of the *tendon sheaths* on the back of the hand,—perhaps as a result of laceration caused by the hyperflexion,—also articular swelling and, more rarely, hyperostosis of one or more metacarpal bones.

The *disorders of sensibility* are generally slight, and in pressure paralysis are in most cases absent altogether. The patient feels some crawling and numbness in the region innervated by the musculospiral nerve. These paresthesias may even precede the onset of the paresis. Objectively, however, we sometimes find a slight decrease of sensation in a circumscribed but rarely in the whole area supplied by the musculospiral. Anesthesia may even be absent in severe injuries and in complete sections of the nerve; but under such circumstances sensory disorders are generally present, and, when the whole area of innervation is involved, are found upon the back of the hand to about the middle, upon the dorsal surface of the thumb, including the lateral parts of the ball, upon the back of the first three fingers, excepting the dorsal surface of the last or both distal phalanges, which are almost always innervated by the median nerve, and upon the forearm, extending in a narrow strip along the extension side (posterior inferior cutaneous nerve). When the nerve is injured where it winds around the arm, this region is generally spared. If the nerve is injured high up, sensation may also be decreased in the external aspect of the upper arm in the region innervated by the posterior superior cutaneous. In a case of complete musculospiral paralysis caused by fracture of the humerus, I found anesthesia only upon the dorsal surface of the first phalanx of the thumb.

The prognosis of musculospiral paralysis is in most cases *a favorable one*. In slight cases of pressure paralysis, in which there is no decrease in electrical excitability, recovery, even without treatment, occurs in several—on an average, in from four to six—weeks, in exceptional cases even in a few days; it may, however, be delayed for several months. Crutch-palsy also tends to recede rapidly. In moderately severe cases, two, three, or even six months may elapse before the normal functions are restored. The chances are less favorable in cases due to knife-cuts, bony fragments, dislocated head of the humerus, etc. If a complete reaction of degeneration is present, the paralysis remains or improves but slowly; recovery before many months cannot be expected. Those cases resulting from constriction are also often severe. When the nerve is completely lacerated or cut through, recovery is only possible if the stumps be reunited, and then only after a long time. Ether paralyses, as a rule, recover in from one to four months.

Concerning **treatment**, see p. 246. As prophylactic measures, all severe and long-continued pressure upon these sensitive nerves should be guarded against, particularly in using Esmarch's or roller bandages, etc. In subcutaneous injections of ether, avoid piercing the fascia, and only introduce the needle after lifting a fold of the skin.

Nerve suturing and *neurolysis* has been done with good results upon the musculospiral nerve (Busch, Wölfler, Czerny, and myself). Monod undertook its suturing after extirpation of a tumor involving the nerve. Sick and Saenger observed paralysis caused by a traumatic defect in this nerve recede after suturing of its peripheral end to the median nerve.

In a case of callus paralysis of the musculospiral nerve, recovery ensued after a fracture occurred at the same place caused by a new injury.

Muscular transplantation (page 158) in old paralyses of this region has been executed with good results.

Many apparatus have been recommended for disturbances in function caused by the flexion position, the flaccid hanging down of the hand and fingers, which are due to defective ability to flex the hand and fingers,—of which that of Heusner deserves special mention: a stiff leather strip encases the forearm and hand as far as the roots of the fingers and holds the wrist-joint in a slight position of extension, the metacarpal of the thumb remaining for the most part free. Upon the back of this strip are fastened four gum strings, which run into broad gum bands, placed around the basal phalanges of the four fingers. These keep the four fingers in a position of extension, without, however, exercising much resistance to the action of the flexors. A more exact description with illustrations is to be found in the *Deutsche med. Wochenschrift* for 1892, page 115.

Credit must be given to E. Remak for exact procedures for the electrical treatment of musculospiral paralyses and the results thereof. He recommends for typical cases stabile cathode treatment : the cathode of a weak, constant current, of about twenty or thirty centimetres square, is placed upon the pressure-point,—above and external to the ordinary place of stimulation for the musculospiral nerve,—and the other pole is placed upon an indifferent point (the sternum). The strength of the current is gradually increased until the patient in attempting to extend the hand feels some ease in its execution. A current strength of from six to eight milliamperes is generally necessary. Remak has shown that the average course is shorter under this treatment. The average length of the paralysis was from twelve to twenty and a half days under this treatment; in cases not treated in this manner, more than thirty days.

PARALYSIS OF THE MEDIAN NERVE.

A paralysis limited to the median nerve is rare, and is almost always of *traumatic* origin. It more often occurs in combination with other brachial paralyses from the pressure caused by luxations and strangulation. The use of Esmarch's bandage may cause an isolated paralysis of this nerve. Injuries due to blows, cuts, or stabs may injure it in its peripheral course. It is seldom involved in fractures of the humerus, more often in fractures of the bones of the forearm, either directly or from the callus. A forcible contraction of the pronator teres is said to be able to cause this paralysis. In one case it was produced by compression of the cervical ribs. Gowers observed it after pronounced distortion of the wrist-joint.

An isolated non-traumatic neuritis of the median nerve occurs rather rarely, though the so-called professional pareses are inclined to localize themselves in this region, symptoms of median paralysis having been observed in tinners, joiners, locksmiths, milkers (Remak), cigar-makers (Coester), carpet-beaters (Reinhardt), dentists, etc. The so-called drummer's paralysis, according to Bruns, may involve the flexor pollicis muscle (and also other muscles of the thumb). The toxic forms of polyneuritis also occasionally involve the median. Puerpural neuritis favors the median and ulnar. Tumors may also develop in the median nerve and cause paralysis of it.

Symptoms.—If the nerve is involved in the upper arm, the following muscles are paralyzed: the *pronators*, the *carpal flexors* (except the flexor carpi ulnaris), the *flexor digiti sublimis* and *profundus* (except the muscle branches for the last three fingers), the *opponens pollicis*, the *flexor pollicis longus* and *brevis*, the *abductor brevis*, and also the *first two lumbricales*. If paralyzed on the distal side of the wrist-joint, only the small muscles of the hand are paralyzed.

The position of the hand is not markedly altered, though it is generally turned towards the ulna, owing to the excessive action of the flexor carpi ulnaris, and on account of deficient pronation is held slightly supinated.

Flexion of the hand is carried on with slight force and with deviation towards the ulnar side. The fingers cannot be properly flexed at the first interphalangeal joints, while flexion of the terminal phalanges is practicable only in the last three fingers. The flexion of the basal phalanges is not involved.

The thumbs approach the index-fingers, are extended, and lie in the same plane as do the fingers; their opposition is lost, also flexion of the terminal phalanx. The patient is unable to pronate the hand, and attempts to replace pronation by rolling the upper arm inward (when the seat of the lesion is high).

Disuse of the two lumbricales does not show itself, in the cases which I have examined, by marked disturbances of function, nor could I detect any alterations in electrical excitability when the interossei remained normal.

The electrical excitability is dependent upon the severity of the lesion (in a similar manner as explained under paralysis of the circumflex). Slight pressure paralysis occurs more rarely in the median than it does in the circumflex; the injury is generally a grave one, and severe alterations in electrical excitability generally accompany it.

Pains, *hyperesthesia*, and particularly *decrease in tactile sensation* are generally present, and the anesthesia may involve the entire area of innervation,—*i.e.*, the volar manus to the fourth metacarpal, the volar surface of the first three, and the radial side of the fourth finger, as also the dorsal surface of the second and third phalanx of the thumb (?), index, and middle finger. Individual variations, often pronounced, frequently occur in the cutaneous innervation on the dorsal surface of the phalanges. Anesthesia, also, may not occur, and even in complete section of the nerves may be confined to a small area. On the other hand, Bernhardt observed in an injury to the median nerve over the wrist-joint marked sensory disturbances, while the motor functions, notwithstanding the reaction of degeneration which was present, were almost intact. At first he ascribed this condition to a partial section of the nerve, but in a later case he sought to explain it by the fact that the ulnar nerve, through a deep branch which anastomosed with the branch of the median innervating the muscles of the ball of the thumb, assumed vicariously the functions of the median.

Vasomotor and trophic symptoms occur more often in traumatic neuritis of the median nerve than of the radial; the skin is, particularly in the later stages, cyanotic, feels cool, and the subcutaneous tissue may be *infiltrated*. *Herpes-* and *pemphigus-like vesicles* develop at times, and leave badly healing ulcers; *glossy skin*, thinning of the terminal phalanges, abnormal growth of the nails, longitudinal striations in the nails, alopecia unguium (where the nerve is imbedded in a cicatrix, this is the only symptom), etc., are other phenomona which occur. *Hyperidrosis* occurs occasionally; more often amidrosis of the volar hand and of the fingers. In one of my cases of partial median paralysis the trophic disturbances extended to the nail of the ring-finger, and the vasomotor symptoms (cyanosis) appeared upon the volar and dorsal surface of the second and third fingers.

The course and prognosis are dependent upon the severity of the lesion, and the discussion in the preceding article will enable one to render a decision on this point, as well as on the **treatment**.

PARALYSIS OF THE ULNAR NERVE.

This is more often observed than is paralysis of the median nerve. It can, in the first place, occur in combination with paralysis of other brachial nerves,—for instance, in luxation paralysis. In its further course it is involved chiefly in connection with the median nerve on the upper or lower arm from injuries, fractures, and their resulting conditions. An isolated lesion of this nerve occurs in fractures of the internal condyle of the humerus, supracondyloid fractures, either from the primary force or from attempts at reposition, but especially when the bony fragment presses upon or bruises the nerve, a laceration resulting very rarely. The *callus* may be the cause of the lesion, either by elevating the nerve from its position and flattening and stretching it, or from it being surrounded by the callus itself. In a woman treated by me the callus paralysis developed two years after a fracture following upon sudden movement. In another case almost two decades elapsed between the injury (with formation of callus) and the onset of the paralysis, which resulted after over-exertion or laceration. In many cases cicatricial tissue was found attached to the nerve. In the forearm the nerve is often incised or completely severed by blows, sabre-cuts, pieces of glass, etc.

Slight pressure paralyses also occur. The nerve can be compressed by a hard support, when the inner surface of the elbow or the condyle region rests upon it. This has been noticed particularly in bedridden, emaciated persons; though I treated a strong laborer, not addicted to alcohol, in whom a mild paresis of the ulnar nerve developed while he was enjoying his noon-day siesta, the elbow of his right arm serving as a prop to his head. The first attack ended in recovery in nine days; a new paralysis, which came on under the same conditions some years later, was likewise mild. Cases of pressure paralysis during sleep have also been observed by Erb, Gowers, and others. Braun refers sleep and narcosis paralysis of the ulnar nerve to pressure exerted by the head of the humerus with the elevated and abducted arm; this is, however, not always the case in sleep paralysis. A luxation and subluxation of the ulnar nerve with the symptoms of paralysis has also been described. A protracted or *forcible flexion* of the forearm is said to have evoked an ulnar paralysis in a few cases. The professional pareses often localize themselves in the region innervated by this nerve. To these belong a paresis observed in glass-blowers, as also in xylographers (Bruns). Something similar is said to occur in bicycle riders.

A *primary spontaneous neuritis* limiting itself to these nerves is very rare, but it has been observed after acute infectious diseases, particularly typhoid fever (Nothnagel, Vulpian, Wolf). In many forms of polyneuritis the ulner takes part in the paralysis. Syphilitic neuritis seems to favor the ulnar, and often involves it alone (Ehrmann, Gaucher, myself). Occasionally a spindle-like protrusion of the nerve-trunk has been noticed. In some cases a syphilitic meningitis of the corresponding roots was the cause of the paralysis. Other tumors may develop upon the ulnar nerve. A traumatic cystic formation was described by Bowlby and Busch. An ascending neuritis was observed by Cenas. Compression from an exostosis-like developmental anomaly on the humerus was noted by Féré.

Symptoms.—In complete paralysis, *the flexor carpi ulnaris, flexor digitorum profundis* for the three last fingers, the *adductor policis*, the muscles of the *ball of the little finger*, the *interossei*, and the *last two lumbricales* are affected.

According to Bardeleben and Frohse, the three lumbricales and the flexor digiti sublimis are occasionally innervated by the ulnar nerve, and, on the other hand, the adductor pollicis at times by the median nerve.

The patient can still flex the hand, but only with radial adduction. He cannot flex the terminal phalanges of the last three fingers nor adduct the thumb; the lost motion caused by paralysis of the interossei and lumbricales is very evident; the basal phalanges cannot be satisfactorily flexed nor the middle and distal phalanges extended. Overaction of the extensor digitorum communis and the long flexor of the fingers causes a claw-hand. This claw position (Figs. 9 and 10) is most pronounced in the fourth and fifth fingers, and decreases from here to the second, because the lumbricales of the first and second fingers, supplied by the median, are still able to produce slight extension of the last two phalanges. *Abduction and adduction* of the fingers are impossible or are reduced to a minimum. The corresponding movements of the little finger are also impossible. In severe cases atrophy comes on, especially pronounced in the interossei and ball of the little finger.

Concerning the electrical excitability, our remarks would not differ from those of previous chapters, so we shall not repeat them here. In slight pressure paralysis it may be entirely normal.

Sensory disturbances are rarely absent in ulnar paralysis. Pains, hyperesthesia, and anesthesia may exist together. Symptoms of irritation are particularly frequent in the cases where a trauma has given rise to conditions favoring a protracted compression. The *decreased sensation*, when the nerve is injured in the upper arm or in the upper part of the forearm, can exist in the entire region innervated by the nerve, —*i.e.*, on the volar surface of the hand, corresponding to the fifth and middle of the fourth finger, the middle finger, and the ulnar side of the ring-finger; the dorsal surface of the hand as far as the middle and the back of the fifth, fourth, and the ulnar half of the third finger. The dorsal surface of the end phalanx of the middle finger is innervated by the median, sometimes also the radial side of the dorsal surface of the end phalanx of the fourth finger.

In a case in which the hypesthesia extended upon the ulnar side almost to the elbow, the median cutaneous nerve was probably also involved.

It should be noticed that the dorsal trunk of the ulnar nerve turns to the dorsal surface as high up as the border between the middle and lower third of the forearm, between the ulna and flexor carpi ulnaris, so that cutting wounds upon the volar surface in the lower third of the forearm do not affect the sensibility of the dorsal hand and fingers. The sensory disturbances are generally distributed over a smaller area than would be expected from the anatomical relations. In complete ulnar paralysis the anesthesia or hypesthesia may be confined entirely to the ball of the little finger or to the entire little finger. In several cases observed by me, a painful sensation of cold in the little finger was complained of. In one the skin of the ulnar region was of a deep-red color. Hesse found decreased temperature upon the skin of the paralyzed finger. Erb described retardation in the conduction of sensibility. In a shot-wound observed by me, sensory and vasomotor disturbances and degenerative atrophy were detected, but no disorder of movement was present.

Trophic disturbances upon the skin occur quite often. Dupuytren's fascia contracture was observed several times (Eulenberg) accompanying a neuritis of the ulnar nerve.

In cases of slight pressure paralysis recovery may follow in a few weeks. In severe cases the course is longer and recovery is often only possible after artificial measures have been instituted (removal of bone-fragments, neurolysis, nerve-suture, etc.), and even then years may elapse before recovery is assured.

There are certain congenital muscle defects in the small muscles of the hand that can evoke the position characteristic of paralysis of the interossei, and fix the hand in such position. The process may, however, recede during the first years of life, as I have observed, probably denoting delayed development of these muscles.

PERIPHERAL PARALYSIS OF THE NERVES OF THE LOWER EXTREMITY.

Diseases of the *lumbar* and *sacral plexuses* are not as common as those of the brachial. The nerves of the lower extremity are also not as often paralyzed as those of the arm.

There have been but few observations of paralysis of the *crural* and *obturator* nerves. The causes were generally tumors which arose from the vertebral column, the retroperitoneal lymph-glands, the pelvis and its contents; *psoas abscesses* can also injure the crural nerve. A *primary spontaneous neuritis* of the crural has been observed twice by me; in one case it was referred to a severe drenching, in the other it was of *gouty origin*. In alcohol neuritis the sensory crural branches are often affected, but a *total bilateral crural paralysis* occasionally occurs in alcoholics. Bruns observed crural paralysis with neuralgic pains in a case of diabetes mellitus, which disappeared under antidiabetic diet; in two other cases it affected the *crural* and *obturator*, and sprang from one leg to the other. An isolated paralysis of the crural is very rarely developed from infectious causes.

The nerve is rather well protected from injuries, though now and then traumatic paralysis of it is observed resulting from direct wounds, fractures of the thigh and pelvic bones, etc.

Gumpertz describes a narcosis paralysis of this nerve; in his case the leg was fixed in a pronounced degree of flexion of the hip for a long time.

I have seen the nerve become paralyzed from the pressure of an *aneurism* arising from the femoral artery.

Symptoms.—1. Paralysis of the *ileopsoas*, which is not present when the nerve is affected outside of the pelvis or after the giving off in the pelvis of the branches for this muscle, the *extensor cruris qudriceps*, the *sartorius*, and the *pectineus*. Total paralysis, therefore, causes inability to flex the hip or to extend the lower leg. In bilateral paralysis the gait is markedly disturbed; in unilateral impairment the patient is compelled to step carefully, avoiding flexion of the knee. The pectineus —which is, according to Bardeleben-Frohse, supplied in some by the crural and in others by the obturator—is not completely impaired in function, and does not cause any distinct disturbance any more than does the sartorius.

2. *Anesthesia* or *hypesthesia* in the region innervated by the anterior and interior cutaneus femoris and the saphenus major, in the anterior and inner surface of the thigh, excepting its upper third (Fig. 24), in the inner surface of the leg, and along the inner border of the foot almost to the great toe.

3. *Absence of the knee reflex.*

Reliable observations concerning the condition of the cremasteric reflex in this paralysis are wanting.

Pressure paralysis, as a rule, begins with symptoms of irritation and with pains, which follow the course of the crural and saphenus. *Muscular atrophy* and alterations of electrical excitability are always present when the nerve disorder is a severe one.

An isolated paralysis of the *obturator nerve* occurs more rarely (compression in difficult labor, pelvic tumors, obturator hernia). It is characterized by paralysis of the *adductors* of the thigh—external and internal rotation is also somewhat impaired—and *sensory disturbances* upon the median side of the thigh in its upper third, sometimes also as far down as the knee.

It has only been lately that we have become acquainted with an isolated disease of the *external femoral cutaneous nerve.* Bernhardt and Roth call attention to the pains, paresthesia, and sensory disorders which occur, and not so rarely, in the area supplied by this nerve. The troubles come on chiefly, sometimes only, in walking and standing; probably because the fascia is then stretched the most. Objectively a more or less pronounced decrease in sensation upon the outer surface of the thigh is noticed, particularly in its lower area. The disorder described by Roth as *meralgia paresthetica*, due to a neuritis of the external femoral cutaneous nerve, occurs chiefly in men. All those whom I treated for this disorder were alcoholics; in one, neuritic phenomena were present in other nerve-trunks. Other factors have, however, been given as causes. It is generally a harmless trouble. In one of my patients it has existed for twenty years, without any other symptoms of disease appearing. It may, however, be a symptom of tabes.

Of the nerves of the *sacral plexus*, the *sciatic* is the most exposed to injurious factors; yet complete paralysis of this nerve is rarely noticed. Almost all movements that evoke sciatic neuralgia (sciatica) can also produce symptoms of sciatic paralysis, which, however, rarely reach a high intensity, but which more often confine themselves to a *degenerative paresis of the peroneal nerve.*

Osseous fragments from fractures of the lumbar vertebræ, sacrum, pelvic bones, *pelvic tumors*, and exudates may compress the nerve-roots. The paralyses occurring *intra partum*, which are generally due to the application of *forceps*, but which can occur spontaneously without any artificial help, and particularly in cases of *narrow pelvis* (Hünermann) —general contracted pelvis—as a result of the pressure exerted by the *head* of the child upon the nerve, are of great practical interest. They generally occurred with occipital presentations, but were observed once in a face presentation and in a case where the head was born last.

It is a remarkable fact that in those cases in which the compression or some other injurious factor involves the nerves in the pelvis, the paralytic symptoms are confined to the peroneal nerve, or at least are most strongly marked in it. It has been assumed that the fibres for the peroneal nerve lie close together in the pelvis, directly upon the bones, being particularly exposed therefore to pressure.[1]

An uncommon high division of both main branches of the nerves has likewise been noticed. Other authors have thought that the fibres for the peroneal are particularly sensitive and less resistant. D. Gerhardt showed that after the death of an animal the extensors of the foot become functionless more quickly than the other muscles; and showed experimentally that lesions which involve the sciatic cause a degeneration of the peroneal first. Perhaps all these opinions contain some truth. It is an established fact that the infectious and *toxic* forms of neuritis involve most frequently the peroneal nerve.

---

[1] Attention is called to the fact that the peroneal nerve for the most part arises from the lumbosacral nerve—*i.e.*, from the plexus branch formed from the fourth and fifth lumbar and first sacral nerves, which lie directly upon the innominate crest, while the rest of the plexus lies upon the pyriform muscle.

Puerperal neuritis can establish itself in the sciatic. Parametritic and septic processes in the pelvis can also extend to the sacral nerves, particularly to the sciatic (K. Mills). In its peripheral course it is exposed to various traumatic influences. Paralysis of the sciatic, complete or partial, can result from subcutaneous ether and corrosive sublimate (antisyphilitic) injections, stretching and other forms of injury, dislocation of the hip-joint, and fracture of the femur.

The *peroneal* nerve is more often paralyzed than is the tibialis posticus. In addition to the etiologic factors already enumerated, the following are of value: in fracture of the fibula the peroneus can be injured directly or by callus. A bilateral traumatic paralysis of this nerve was due in one of my cases to a severe injury, in which both legs were hit by a flying rod. It can be caused by laceration in running and by missteps (Charcot, Remak). I saw it occur in an alcoholic simply from a forcible extension of the leg.

A single observation has been made of peroneal paralysis from the application of Esmarch's tourniquet (Wiesmann), and one from the pressure of stilts (Bergonié). Gerhardt observed it develop in a man who slept with crossed legs.

In laborers who work in a stooping position,—potato-pickers, asphalt-workers, seed-sowers, etc.,—a peroneal paralysis may develop (Benker, Roth, Bernhardt, Hoffmann). It is probably due to pressure exerted between the tendon of the biceps muscle and the head of the fibula.

Paralysis of the *tibialis posticus* may be of *traumatic* or of *toxico-infectious* origin. In a child who had fallen through a cane-chair and remained hanging by the hock of the knee in the frame, I observed a paralysis of this nerve which did not recede completely. In seed-sowers a paralysis of the peroneus and tibialis posticus can develop (Hoffmann). In one of my cases it confined itself to the tibialis posticus. Polyneuritis involves these nerves rather frequently. In a case of dermato-myositis which I saw, the disease extended to the tibialis posticus nerve, at the same time producing marked edema of the hock of the knee.

Symptoms of Peroneal Paralysis.—The *extensors* of the *foot* and of the *toes*, the *abductors*, and the *tibialis anticus* among the adductors are paralyzed. The foot falls from its own weight, remains in an equino-varus position, cannot be raised, nor can the first phalanx of the toe be extended. If the paralysis continues the foot becomes fixed in a position of "foot-drop" from contraction of the triceps suræ. Contractures may also come on in the interossei, fixing the basal phalanges of the toes in a position of flexion. Walking is difficult, and the toes scrape the floor, unless the patient overflexes the leg at the knee and hip. The foot touches the ground first with its outer border and tip. The paralysis is generally a degenerative one.

In neuritis of the peroneal nerve the paralysis may be a partial one; for instance, not involving the tibialis anticus or the peroneus longus muscles for a long time.

If *anesthesia* be present it is found in a small area on the anterior surface of the leg, over the crista tibiæ and outward from this (in the lower half), and also upon the dorsal surface of the foot and toes, not, however, upon the outer and inner borders of the foot. (See Figs. 24, 29, and 30.) Individual variations, however, occur (Frohse).

Vasomotor and trophic disturbances are sometimes present.

Paralysis of the *tibialis posticus* is characterized by loss of the *plantar flexion* of the foot and toes; if the popliteus is involved, inward rotation of the flexed leg is hampered. Paralysis of the interossei can produce a claw position of the toes (*pied en griffe*); separation of the toes is also hindered, though no disturbance of function of importance is produced thereby. The patient cannot lift himself upon the tips of his toes. Walking is very difficult. The overweight of the antagonists can evoke a pes calcaneus or valgus.

Sensation is decreased or lost upon the posterior inner surface of the leg, upon the outer border of the foot, the sole, and the plantar surface of the toes.

If the sciatic is involved *in toto*, the flexors of the leg are paralyzed in addition to the peroneus and tibialis posticus. According to Bardeleben and Frohse it also occasionally innervates the adductor magnus. The disturbance of gait is pronounced, but may be lessened by appropriate supporting apparatus. If the nerve is entirely divided, nerve-suture may be necessary, though it has been done only a few times upon this nerve (Dallas and Prath).

Isolated paralysis of the *glutei nerves* is very rare. It is not uncommon in conjunction with the sciatic in diseases of the sacrum and pelvis—fractures, tumors, caries with abscess, as in a case of Laehr. Paralysis of these nerves causes paresis and atrophy of the glutei muscles, the pyriformis and tensor fasciæ latæ. Abduction and inward rotation of the thigh, and particularly extension movements of the hip-joint, as in climbing stairs, etc., are hampered.

PERIPHERAL PARALYSIS OF THE CRANIAL NERVES.

The diseases of the first and second cranial nerves are so allied to cerebral disorders that they will be discussed in connection with them.

PARALYSIS OF THE NERVES OF THE OCULAR MUSCLES

will be discussed here only as far as the nerves are involved in their extra-cerebral course at the base of the brain or in the eyeball, though the border-line between these and central lesions (see Ophthalmoplegia) is not sharply defined.

These paralyses are often accessory symptoms to a *general disease*, or to one of the *central nervous system*, and are, further, often *secondary*, —*i.e.*, produced by the extension of a process in the vicinity, and are rather rarely *primary*. Cold is an etiological factor. *Rheumatic* paralysis of the ocular muscles is naturally more rarely diagnosed than formerly, since it has been learned that isolated paralysis of ocular muscles is a frequent forerunner of central lesions, and a frequent result of infectious diseases. Notwithstanding, we insist that a unilateral paralysis of the abducens and oculomotor or of some of its branches may result from exposure to cold, which after an acute course soon recovers. A complete unilateral ophthalmoplegia of rheumatic origin has also been described and referred to an inflammation near the superior orbital fissure. It is probable that the rheumatic paralysis is a neuritis, as many observations have shown the frequency with which the ocular nerves are involved in multiple neuritis.

The ocular paralysis is often of *infectious* origin. The most common is the *post-diphtheritic*. It involves most often the muscles of accommodation on both sides, and not rarely the branches for the external ocular muscles, the abducens more often than the oculomotor; but it may affect the whole third nerve or all external ocular muscles. A trochlear paralysis under these conditions is rarely observed (Krauss). Even though some of these paralyses are of nuclear origin (see Paralysis Postdiphtheritica), alterations of the nerve-trunks (for example, hemorrhage and degeneration) have been observed. In the course of and as a result of *influenza*, the ocular muscles may become paralyzed, the ciliary muscle, sphincter pupillæ, and also the external muscles. Only in some cases (Michel, Bunzel) was it observed in *acute rheumatism*, also in a few cases after *scarlatina* and *pneumonia* (Mauthner, Wadsworth, etc.). It may develop during an attack of typhoid (Ebstein).

I observed an oculomotor paralysis develop in the course of an *acute nephritis*. *Syphilis* is a frequent cause of this paralysis, generally gummatous tumors or a basal gummatous meningitis. A primary *gummatous neuritis* of the ocular nerves also occurs; perhaps also a primary syphilitic atrophy. Finally, *exostoses* may involve the ocular muscles, as may also *syphilitic periostitis* of the base of the skull or of the orbit, or *tuberculosis*. *Thrombosis of the cavernous sinus* may also be a cause.

Although we know a number of poisons that can influence the nerves of the pupil and of accommodation, we have little definite knowledge concerning the *toxic origin* of palsy of the ocular nerves. There is no doubt that a *chronic alcoholic intoxication* may paralyze the ocular nerves and produce a neuritis of them (nuclear paralysis of the ocular muscles of alcoholic origin will be spoken of separately). A paralysis of the ciliary muscle and other ocular muscles occurs rather often during diabetes. This paralysis is rarely due to gout (Galezowski). Saturnine intoxication involves the ocular nerves very rarely. Fish-, meat-, and bologna-poisoning paralyzes first the ciliary muscle, then the sphincter pupillæ and the external ocular muscles. (Observations of Cohn, Leber, Guttman, Scheby-Buch, etc.) Accommodation paralysis and mydriasis have also been referred to poisoning by oysters and to auto-intoxication from the intestines (Panas). In these types of ocular paralysis we are probably dealing with nuclear diseases of a functional or organic nature.

Compression of the nerves from tumors, aneurisms, meningitic exudates, hemorrhages, bony fragments, periostitic deposits, inflammation of the orbital connective tissue, etc., is a common cause of these paralyses.

Lesions of the orbit and cranium (basal fractures) injure the ocular nerves not only from the fracture and accompanying hemorrhage, but contusions without a separation in continuity of the bones may directly paralyze them, probably from the fact that they produce hemorrhages in the nerves, a predisposition existing thereto.

Paralysis of the ocular nerves rarely occurs through pressure of the forceps in labor. *Arteriosclerosis* of the basal arteries may directly compress the ocular nerves, or the tortuous course of the main artery may cause a stretching of its branches and thereby crush and constrict the nerves passing over them. This, however, rarely occurs.

It is seldom that you see a *primary hemorrhage of the ocular nerves.* I saw a trochlear paralysis suddenly appear in a young, non-syphilitic man, who had profuse nose-bleeds accompanied by vertigo and apoplectic symptoms. It gradually disappeared.

Sänger observed hemorrhages of the oculomotor nerve in tubercular meningitis.

Ocular paralysis occurs in tabes, multiple sclerosis, cranial tumors, etc., and diseases of the pons and corpora quadrigemina. Only rarely is the tabetic ophthalmoplegia due to peripheral neuritis, as in a case observed by Dejerine and Petreen.

Finally, there is a *congenital* and *hereditary* form of ocular paralysis, which may involve some branches (levator palpebræ superioris, rectus superior, of one or both sides), or, more rarely, all the branches of the nerves supplying the external ocular muscles (Gräfe, Mauthner, Möbius, Kunn, etc.). We do not know anything positive concerning its anatomical basis. Hencke has found an absence or degeneration of the muscles, and Siemerling observed nuclear atrophy in a case of congenital ptosis. I saw several cases in which a congenital paralysis of the ocular muscles existed with other developmental anomalies.

Symptoms.—The signs of an abducens and trochlear paralysis have been already described. (Pages 80, 81.)

Total oculomotor paralysis presents the following symptoms: *ptosis*, paralysis of the rectus superior, inferior, internus, inferior oblique, and of the sphincter pupillæ and the muscle of accommodation.

The upper lid hangs down so as to hide the pupil. The lids can be moved very slightly by contraction of the occipitofrontalis, but not at all if the eyebrows are held fixed. The eyeball can only be moved outward, and any attempt to move it draws it downward and outward. Gradually the eye is brought persistently into the outer palpebral fissure, due to secondary contracture of the rectus externus. The pupil is of average size and does not react to light; atropine dilates it more. In attempts at convergence it does not contract. Illumination of the normal eye has no influence on the pupil of the paralyzed one. The eye is easily dazzled.

Paralysis of the external muscles occasionally produces a slight *exophthalmos*.

Diplopia is present in the whole field of vision provided the upper lid is raised, and is accompanied by the disorders peculiar to this condition. If ptosis is absent, the patient shuts his eye spontaneously to prevent the diplopia; this closure of the lid may, as I observed in one case, produce a facial spasm. *Pains* at times accompany the oculomotor paralysis, especially if it is a rheumatic or compression paralysis.

It is not possible to make an electrical examination of the muscles innervated by the oculomotor nerve nor of the nerve itself, though Salomonsen claims to have produced a sluggish contraction in degenerative paralysis of the levator palpebræ superioris with direct galvanic stimulation.

In partial paralysis of the third nerve the movements are only limited; it often occurs that some branches are paralyzed while others are only paretic. A *partial oculomotor paralysis* is more often observed, in which the rectus superior and levator palpebræ superioris, or one of the other recti, or all the external ocular muscles with the exception of the levator palpebræ superioris, or the internal rectus, or only the muscle of accommodation in combination with or without the sphincter iridis, are involved.

As a rule, in diseases of the nerves of the ocular muscles, *all* the muscles are completely or partially paralyzed, while in nuclear affections, unilateral or more often bilateral paralysis of some of the muscles occurs, the rest remaining normal.

A bilateral paralysis of the sphincter pupillæ and muscle of accommodation points, therefore, to a nuclear affection. This also is probably the condition when, in complete bilateral paralysis of all the external ocular muscles, the muscles of accommodation functionate normally. The distinction is not always a positive one, as when a paralysis of some branches (for example, the sphincter iridis) remains as a residuum of a total peripheral (basal) oculomotor paralysis. I have often seen this in syphilitic cases. The sphincter iridis can also remain uninvolved in basal diseases of the oculomotor without (with Adamük) suggesting an abnormal course of the pupillary fibres. Dammron and Meyer (also Dejerine) noticed the presence of a peripheral neuritis of the ocular nerves in a case in which the paralysis of the ocular muscles resembled nuclear disease.

An involvement of the neighboring cranial nerves points to a peripheral seat of the paralysis.

Disease of the ocular nerves themselves never produces associative paralysis, though a paralysis of all except the levator palpebræ superioris was observed a few times (Thomsen, Ormerod) in basal disease of the oculomotor.

Fuchs and Silex described a progressive paralysis of the levatores palpebræ superioris occurring in old age, which they believed to have been a primary myopathy of these muscles. Hereditary ptosis also occasionally develops only after birth.

The course and prognosis depend upon the cause and the character of the original disease. The so-called rheumatic cases and those developing after the acute infectious diseases generally recover, as a rule, within a few weeks. Those following influenza may be very stubborn. The syphilitic form in general has a good prognosis. If it has not lasted long, so that atrophy has not taken place, a correct treatment will cause complete or partial restitution. In injuries the prognosis depends upon the severity of the lesion. The prognosis in non-syphilitic tumors is unfavorable, as basal tumors are almost never operable.

Ocular paralysis occurring in multiple neuritis almost always disappears when the original trouble recedes.

Ocular paralysis occurring in tabes has a tendency to retrograde in the first stage, though it may be permanent, or even progressive, in character. A symptom of grave import is an *isolated reflex pupillary rigidity*. It is generally a prodrome of tabes or dementia paralytica. It may also be the only sign of a *syphilitic cerebral disease*, and occasionally may be a symptom of chronic alcoholism. A spontaneous *paralysis of accommodation* combined with mydriasis, and generally, also, with loss of the light reflex, is also of grave import.

I have seen cases of this kind in which only after five or ten years did other symptoms of tabes or paralysis occur, though sometimes this paralysis does not precede a severe disease. I had a female patient with pupillary rigidity which was probably a result of cerebral lues, whom I examined fifteen years later without finding any new symptoms.

Treatment.—In syphilitic cases the proper treatment must be rigidly enforced. According to my experience, a *sweat-cure* is the best agent in rheumatic, infectious, and toxic cases. In traumatic and rheumatic cases, local *antiphlogosis* and *bloodletting* are in place; *vesicants* (cantharides plaster to the back of the ears) may also be tried. In diabetes, gout, etc., proper *dietetic* rules must be enforced.

Electrical treatment of the ocular paralyses is not of much benefit, but it may be used when the cause cannot be removed. A galvanic treatment is the best, the cathode being placed on the closed eye, the anode on the back of the neck. Both electrodes may also be placed on the temples or in the neighborhood of the paralyzed muscles,—for example, in paralysis of the rectus superior, in the frontal region over the eyes, etc. The current should be strong enough to contract the facial muscles. Duration, two to three minutes. Faradic treatment is not in favor. It is best to prohibit entirely all attempts to stimulate the muscles directly by fine electrodes introduced into the conjunctival sac.

Subcutaneous strychnine injections may do good in some cases.

To avoid the disturbances arising from the diplopia, it is advisable to order glasses which, by means of a ground glass, prevent all use of the affected eye. The eye may also be covered by a shade. The use of strong prisms to unite the double pictures is not advisable. When the condition is slight, weak prisms may be used. Gymnastics are of doubtful therapeutic benefit; an object is slowly brought into the field of double vision from that of single vision, the patient endeavoring as long as possible to keep the single object in view.

Regarding the *operative treatment* of ptosis and paralysis of the ocular muscles, consult text-books on eye diseases.

The patients often endeavor themselves, by various methods, to lift the paralyzed eyelids. A young man, whom I treated, suffering from ptosis duplex, fastened to his glasses a protruding stirrup, which pressed from below upon the lid in such a manner as to elevate it. Figs. 161 and 162 illustrate the effect of a similar mechanical appliance used by Heckel in cases of ptosis.

FIG. 161.

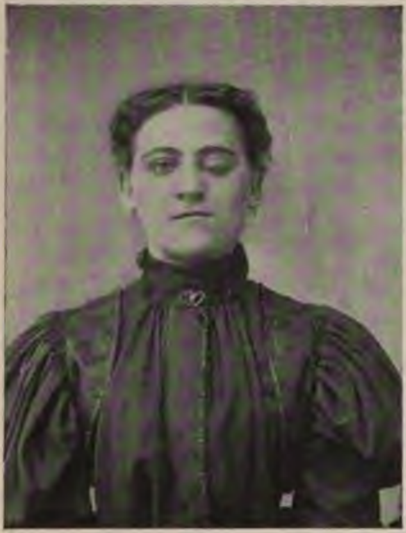
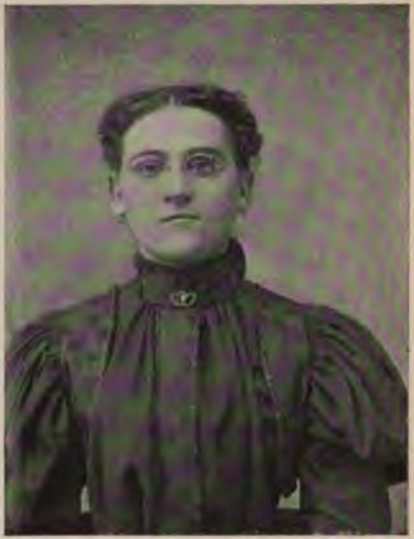


FIG. 162.



PERIODICAL PARALYSIS OF THE OCULOMOTOR NERVE.

Cases have been observed (Gubler, Camuset, Saundby, Moebius, Senator, Pflüger, Vissering, Charcot, Manz, and others) in which there occurred from time to time, generally at regular intervals, a paralysis of the oculomotor nerve, which again disappeared after some days, weeks, or months. *Children* were generally affected; a *nervous* predisposition was not noticed. There occurred with the paralysis, which generally attacked the same nerve (not passing from one side to the other), *headache* or pain in the eyeballs, the forehead, or the whole front of the head on the side of the paralysis, with nausea and vomiting. The headache generally had the character of *migraine attacks*, returned every four weeks or at longer intervals, and was almost always accompanied by the oculomotor paralysis. It is the rule that this begins the attack, and disappears when the other symptoms come on. In contradistinction to typical migraine, the headache and the vomiting may last for a week. This paralysis involves generally the whole oculomotor nerve; in some cases some of the branches were spared. The paralysis may even confine itself to the levator palpebræ superioris (Knapp). A decrease in sensation of the region supplied by the *first branch of the trigeminal* was observed in some cases. Karplus saw the *second* branch also involved. In a case examined by me, I found that *albuminuria* was the only objective symptom which complicated the disorder, though the subjective troubles indicated neurasthenia.

There are cases of *purely periodical* and of *periodical exacerbating* (Senator) oculomotor paralysis. In the latter type a paresis of the nerve or of some of its branches, which may increase to total paralysis, is present between the attacks; in the first we find nothing in the intervals. It also occurs that the paralysis disappears after the first attacks, but persists partly later on.

We do not know anything positive concerning the cause of these symptoms nor the seat of the disease. Some, as Möbius and Brissaud, assume a nuclear disease; others, and the majority, a basal disease. In the cases which came to an autopsy (Gubler, Weiss, Thomsen-Richter, Karplus), the trunk of the oculomotor was found diseased; in one, a plastic *exudate*, in the others a *neoplasm* (tubercle, fibrochondroma, neuroma) was found. Many views are expressed—functional disturbance, local hyperemia, vascular anomalies, and the like—to account for the symptoms. The most plausible is that of Charcot, that periodical oculomotor paralysis is allied to *hemicrania,* and probably the latter, also, can be referred to *vasomotor* influences. A vascular spasm inhibits the flow of blood to the nerves, and thereby produces the paralysis; or the arterial nerves become paralyzed, and the overfilling of the vessels produces a corresponding compression of the nerves.(?)

These attacks may recur many times without injuring the nerves. But in time a *degenerative* and inflammatory process takes place, which is not capable of complete retrogression.

It should also be understood that a circulatory disturbance recurring like this one can be the starting-point for exudative processes and neoplasms.

Charcot emphasizes its intimate connection with migraine, and speaks of *migraine ophthalmoplégique.* Karplus is opposed to this. Cases of hemicrania have been observed (Massalonge, myself, and others) in which a temporary paralysis of single ocular muscles occurred (levator palpebræ superioris, sphincter pupillæ, etc.).

The course is progressive or retrogressive, or may come to a standstill. Only progressive cases can have a bad **prognosis.** Prognosis *quoad vitam* is good, if we exclude the cases which are caused by a neoplasm.

The treatment is similar to that of migraine and of paralysis of ocular muscles.

PARALYSIS OF THE TRIGEMINAL NERVE.

Anatomy.—The *trigeminal* arises by two roots from the pons or the ventral surfaces of the middle cerebellar peduncle. The anterior one is small and contains only motor fibres; the posterior one is much larger, and is purely sensory. They lie side by side, and in such a manner that motor fibres run on the lower median side of the sensory, and pass through a cleft of the dura on the apex of the petrous bone, on the side of the exit of the abducens nerve, to a cavity on the upper surface of the petrous pyramid. This cavity is formed from the dura mater, is called the *cavum Meckelii*, lies lateral to the sinus cavernosus, and extends from the petrous bone anteriorly and slightly laterally to the median angle of the superior orbital fissure, the foramen rotundum, and the foramen ovale. In this space is formed the posterior root of the Gasserian ganglion, from which arises the third branch of the trigeminal, while the anterior one passes below the ganglion, and only joins with the third branch on the other side of the ganglion. According to the latest researches, the sensory trigeminal root arises from the Gasserian ganglion and passes to the pons, and from here descends into the spinal cord, to form the spinal roots (formerly called the ascending). It therefore degenerates downward (Sherrington, Biedl, Bastianelli).

Sympathetic fibres run alongside the first branch, the ramus *ophthalmicus*, which innervate the dilator pupillæ (and non-striated muscles of the lid), having passed to the eye or the ciliary ganglion.

The second branch, the ramus supermaxillary, is connected with the *sphenopalatine ganglion* through the *sphenopalatine nerve*, which is connected with the *facial* at the geniculate ganglion by the Vidian or superior petrosal major nerve.

The third branch is connected with the otic ganglion. From this arises the smaller petrosal nerve, which sends a branch to the facial and connects with the tympanic nerve, or Jacobson's, in which it is continued with the glossopharyngeal nerve.

Fine fibres connecting the otic, sphenopalatine, and Gasserian ganglia have been noticed.

The first and second receive only sensory, the third branch also motor fibres.

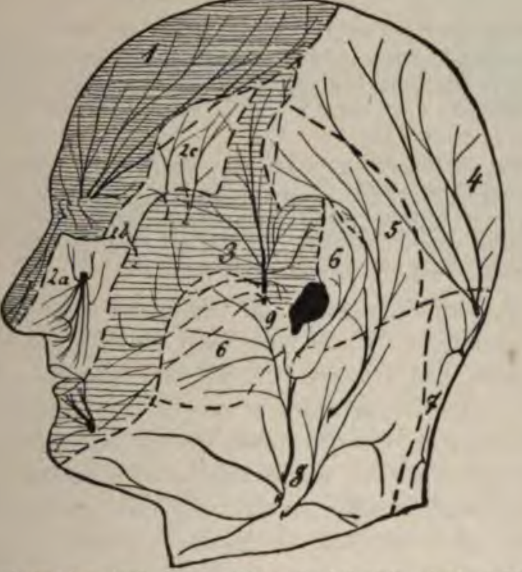
The first supplies the skin of the head in the region marked 1, Fig. 163, from the palpebral fissure to the vertex; also the conjunctiva, cornea, iris, the mucous membrane of the frontal sinus, and a part of the mucous membrane of the nose. It also has *secretory* fibres for the tear-glands, which, however, according to recent conceptions (Goldzieher, Jendrassik), arise from the facial, which leave the greater superficial petrosal with it, and pass through the sphenopalatine ganglion into the ramus orbitalis.

The second supplies the skin of the face between the eye and the mouth (Fig. 163, 2*a*, 2*b*, 2*c*), the mucous membrane of the upper jaw, the nasolachrymal duct, a part of the nasal mucous membrane, part of the gums, the middle ear, and the antrum of Highmore. It also contains sensory fibres for the upper teeth, and probably, also, taste-fibres.

The third branch innervates the skin of the lower jaw, the external ear, and the temporal region, marked 3, Fig. 163, the mucous membrane of the tongue, the lower jaw and the cheek, the lower lip, and the lower teeth. It also contains motor fibres for the masticatory muscles, the tensor tympani, the sphenostaphylinus, the mylohyoid, and the anterior belly of the biventer digastric (biventer).

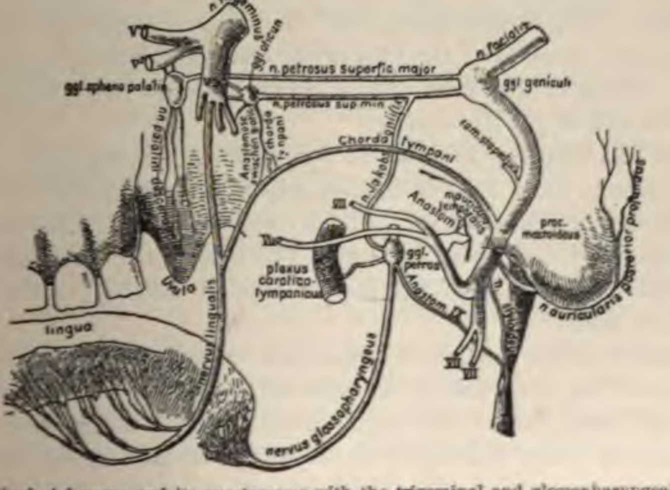
Lately, particularly through the researches of Frohse and Zander, it has been shown that great variations exist in the innervation by the trigeminal. Some places are alternately innervated by different branches, others are supplied at all times by two or more branches. Krause's observations confirm this. This nerve has but slight influence on the pupils. We are not yet certain as to the course of the *taste-fibres.* (Compare Fig. 164.) Those for the anterior two-thirds of the tongue arise in the chorda tympani; those for the posterior part and gums, in the glossopharyngeus. The further centripetal course of these fibres is not known.

FIG. 163.



1. First branch of the trigeminal. 2. Second branch of the trigeminal. 3. Third branch of the trigeminal. Area of the first and third branches of the trigeminal marked by parallel lines; area of the auricular ramus of the vagus in the concha of the ear marked black. 2a, infraorbital; 2b, zygomatico-facial; 2c, zygomatico-temporal; 3, auriculo-temporal; 4, occipital magnus; 5, occipital minor; 6, auriculo-magnus; 7, cervical post. (dorsal); 8, cervical lateral (ventral); 9, auricul. vagi.

FIG. 164.



Course of the facial nerve and its anastomoses with the trigeminal and glossopharyngeal nerves. (After Leube.)

According to Schiff, Erb, and others, the fibres of the chorda tympani reach the second branch of the trigeminal from the facial through the great superior petrosal and the sphenopalatine ganglion. Others, as Ziehl and Müller, regard them as reaching the third branch of the trigeminal through the small superior petrosal and the otic ganglion.

Cases of central disease of the trigeminal without any disorder of taste have been observed. According to others (Brücke), the chorda fibres join the trunk of the glossopharyngeal through the small superficial petrosal and Jacobson's nerve, so that this must be regarded as the nerve of taste. Landois believes that they reach the glossopharyngeal in another way.

This last theory is strongly opposed to one of Gowers and Wallenberg, who regard not only the chorda fibres but also the taste-fibres of the peripheral glossopharyngeal as joining the trigeminal through the small superior petrosal. This conception is, however, contradicted by certain observations (Bruns and others). Other theories, as those of Lussana, Schulte, Stich, etc., will not be discussed here.

Primary isolated diseases of the *trigeminal* nerve are rare, if we exclude the neuralgias. Cases have been observed by Müller, Archer, Ferrièr, Schmidt, and Gowers. A primary neuritis of these nerves rarely occurs. Hirschl described a case of paralysis of the entire trigeminal as rheumatic. Gruber also called his case rheumatic, in which a paralysis of the facial and motor trigeminal occurred in the course of an otitis. Polyneuritis rarely involves the fifth nerve and its branches. It is, however, often involved in disease-processes at the base of the brain, especially those which arise in the middle and posterior cranial fossæ. The branches may also be injured in their peripheral course through the superior orbital fissure, the sphenopalatine fossa, the inferior orbital fissure, or in the upper and lower jaws.

Tumors and *chronic meningitic* processes are especially liable to involve the trunk of the trigeminal and the Gasserian ganglion or its branches. This is particularly the case with gummatous meningitis. A *gummatous neuritis* of the fifth nerve has been observed a number of times. *Caries* of the basal cranial bones, especially the sphenoid, often causes disease of the fifth nerve.

The first branch is involved in tumors which develop in the superior orbital fissure, which arise in the *hypophysis,* in aneurisms of the *internal carotid,* and in orbital tumors (in one case varix of the sinus was found to be the cause of the compression). The second and third are affected by tumors on the floor of the middle cranial fossa and of the sphenomaxillary fossa.

Diseases of the peripheral trigeminal are often of *traumatic* origin. Injuries of the orbits and of the base of the skull may be a cause. The peripheral branches of this nerve may, however, be involved in any facial injury. The fifth nerve is often injured in surgical operations, resections, an extraction after Thiersch, or in extirpation of the Gasserian ganglion.

It should be borne in mind that the trigeminal is not rarely involved in diseases of the brain and spinal cord, epecially in tumors, hemorrhages, and softening of the pons and oblongata, as well as in tabes, syringomyelia, etc. In diseases of the pons the sensory and motor portions may become involved, while in diseases of the spinal cord, as a rule, only the sensory root is affected.

Symptoms.—If the *trigeminal trunk* is involved *in toto*, we find disturbances of sensibility and motility, secretory (?) and often also trophic symptoms.

The anesthesia extends over the whole area of the skin innervated by this nerve, over the mucous membrane of the conjunctiva, cornea, nose, mouth, tongue, cheek, and gums. The observations of F. Krause, however, show that the anesthesia which follows a complete disturbance of the fifth nerve does not cover such a large region as had heretofore been thought. The secretion of tears is impaired upon the side of the disease. This, however, seems to result only when the nerve is involved in the sphenopalatine ganglion or in its peripheral part.

The nasal and lachrymal secretions are defective, causing a dryness of the mucous membranes. The sense of smell is thereby impaired. Krause believes with Magendie that the trigeminal assists in the sense of smell. The anæsthesia of the conjunctiva which is present produces loss of the lid-reflex. The corneal reflex is likewise absent, also the reflex for sneezing and for the palate. The mucous membranes are insensitive to ammonia and similar pungent substances. A glass placed between the lips is felt only on the healthy side, and feels as if it were broken. The patients frequently bite their cheeks.

The *gustatory* disturbance varies greatly. In some cases this sense is completely absent; in others it may not be affected at all or only in the anterior two-thirds of the tongue.

Herpes is the most common trophic disturbance occurring in trigeminal paralyses, though it seems to accompany only a certain form of neuritis. Neuritis of the ramus ophthalmicus may produce a very *painful zoster ophthalmicus*, which may involve the eye itself.

The so-called *neuroparalytic keratitis* commences with a cloudiness, followed by ulceration, perforation, and, finally, inflammation of the whole eye. Formerly it was thought, with Magendie, that this could be referred to the anesthesia and the liability of injuries occurring through the absence of sensation. The fact that it was absent if the eye was protected by a shade from foreign bodies, seemingly proved this. But the keratitis is often absent even in complete anesthesia, and protection of the eye in other cases did not prevent the condition from arising.

Meissner believed that it was due to the section of certain fibres which he claimed to be trophic fibres. But this is denied by Senftleben. Others claim that only lesions of the ganglion and the roots coming from it produce the keratitis (Gaule). The latest conception, which Samuel already believed, is based upon the experiments of Turner and the experimental observations by Krause. According to these it is not the paralysis of the trigeminal or the loss of trophic influences, but the *irritation* accompanying the inflammation which produces the keratitis; it is, therefore, not a neuroparalytic, but a neuritic affection. The anesthesia of the cornea makes it less resistant to injuries, so that traumatic disease heals slowly, but section of the trigeminal does not produce trophic disorder (see page 71). The fact, that this corneal disease is found also in neuritis and in compression of the nerves through tumors, etc., confirms this.

Other trophic changes that are noticed are ulceration of the mucous membrane of the cheek and nose, etc., and spontaneous falling out of the teeth, which, however, occurs almost entirely in central lesions (tabes).

It is doubtful whether the so-called hemiatropia facialis is due to disease of the fifth nerve. *Motor* symptoms only appear in diseases of the *anterior root* as well as of the third branch of the nerve. It is injured in extirpation of the Gasserian ganglion. Paralysis of the motor fifth involves the masticatory muscles—the *masseter*, the *temporal*, and the *pterygoids*. Paralysis of the masseter and temporal may be recognized by the fact that in chewing, the mouth being tightly closed, these muscles do not contract. This can be told by palpation with the fingers.

Peripheral paralysis of these muscles is accompanied, as a rule, by alterations of electrical excitability, decrease of the same, or reaction of degeneration. Atrophy may occur later on. The lower jaw, in unilateral paralysis, can be moved only to the injured side, and in opening the jaw deviates somewhat from this side.

Paralysis of the digastric and mylohyoid does not produce any noticeable symptoms, though the floor of the mouth feels somewhat more flabby than on the sound side. Paralysis of the other muscles need not concern us here, as no characteristic symptoms are produced thereby. In bilateral paralysis of the motor trigeminal the mandibular reflex is lost.

Some cases of unilateral atrophy of the masticatory muscles develop spontaneously (Remak, Werner).

The above refers to *complete* paralysis of the trigeminal, as is seen in complete division of the nerve. In *simple compression*, the paralytic symptoms are incomplete and combined with symptoms of *irritation*—neuralgic pains, hyperesthesia, etc. Then follows a hypesthesia, particularly decreased sensation for touch and pain, which is only gradually succeeded by a complete loss of sensation.

Stimulation of the peripheral sensory branches of the trigeminal may produce facial spasm, cough, and vertigo, through reflex action.

We find in anesthesia of the trigeminal region a certain decrease of the movements of the facial muscles, which, however, never amounts to paresis. This corresponds to the experience of Filehne and Exner concerning the so-called sensomobility.

The course and prognosis depend upon the character of the original disease and upon the severity of the trouble. Hirschl and Gruber observed a retrogression of what they considered a rheumatic affection of the fifth nerve.

The treatment consists in combating the cause. Never exclude syphilis except when antisyphilitic treatment is found useless. Open all abscesses and extirpate all tumors, etc. Use narcotics for the pain, and electrotherapy. As a prophylactic measure when there exists an anesthesia of the cornea and conjunctiva, guard against the entrance of foreign bodies into the eye.

FACIAL PARALYSIS (PROSOPOPLEGIA)

is the most common neural paralysis. It may be peripheral or central. It is a peripheral paralysis when the nerve-trunk is involved after its exit from the pons, in its intracranial or further course, in the internal auditory meatus, in the Fallopian canal, after its exit from the stylomastoid foramen, or if the peripheral branches are affected.

Exposure to cold is given as one of the chief **causes** of facial paralysis (*rheumatic* or *refrigeratory* facial palsy). There is no doubt that exposure to cold (draft, sleeping beside an open window) is the cause of a great number (seventy-three per cent.) of the cases in otherwise normal individuals. The latest observations make it probable that an *infectious* process, which produces a neuritis, is often a factor. Minkowski, in an anatomical examination of a case of this character, did not find any inflammatory alteration of the neurilemma, but only a simple degeneration. This, however, does not speak against its infectious character.

Diseases of the middle ear and *caries of the petrous bone* may easily involve the facial. This nerve lies so near the tympanic cavity, and is separated from it by so thin a lamella of bone, that the inflammation easily passes to it. Facial palsy may result from an otitis, and both these diseases may be caused by the same disease; for example, in exposure to cold, influenza, typhoid, etc. In an autopsy by Darkschewitsch and Tichonow, on a case of otitic facial paralysis, a simple neuritis of these nerves was found, which had no connection with the carious process. It is often injured in caries of the petrous bone. Gowers saw the paralysis follow a *tonsillar angina*. Hatschek observed it after mumps. It occurred once after articular rhumatism with erythema multiforme. It is often combined with herpes zoster.

Gout, *diabetes mellitus*, the *puerperium*, *diphtheria*, *leukemia*, and especially *syphilis*, are reckoned as causes. I observed facial paralysis follow a *mastitis*.

Syphilis generally attacks the facial nerve at the base of the brain, a basal gummatous meningitis or a gummatous tumor affecting it. The syphilic processes may also involve it inside of the Fallopian canal. It is noteworthy that a facial paralysis, probably of a *neuritic* character, may develop in the *early stages of syphilis*, a few months after the primary infection (Boix, Goldflam, and others).

Other diseases, as *meningitis*, *neoplasms*, and *aneurisms*, which form at the base of the skull, may involve the facial.

It may also be injured by traumata at any place in its course. The paralysis following upon the extraction of teeth has an unknown cause (v. Frankl-Hochwart).

In a case of Stocquart, an inflammation extended from the wisdom-tooth to this nerve.

Polyneuritis, especially alcoholic paralysis, may also involve the facial and produce a *bilateral peripheral paralysis* of it.

Diplegia facialis is observed in diseases of the pons and medulla, aneurisms, syphilitic basilar meningitis, rarely in bilateral diseases of the petrous bone and middle ear, and occasionally in rheumatism (Romberg, Mott, Stintzing, and myself). (See Fig. 167.) A facial paralysis occurs sometimes in connection with tetanus.

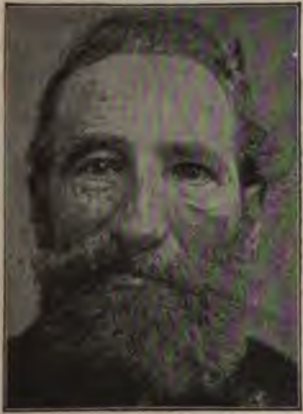
Facial paralysis may be *congenital*, or occur *intra partum* from the use of the forceps, etc. This latter form may be bilateral (Seeligmüller, Edgeworth). In one case (Vernier) it occurred in a breech presentation with early rupture of the fetal membranes. *Congenital* facial paralysis is generally bilateral and combined with ocular paralysis (Möbius, Bernhardt); it is rarely confined to one side (Stephan, Schultze). It may spare some muscles; for instance, the orbicularis oris. Remak described a unilateral paralysis which confined itself to the platysma and chin muscles.

We do not know anything definite concerning the anatomic basis of these congenital facial paralyses. Möbius regards it as a congenital nuclear atrophy.

A *neuropathic predisposition* is of moment as an etiologic factor (Neumann). Charcot saw it occur in a number of sisters. I treated a patient in whom the paralysis resulted from fright, and returned years later after another shock.

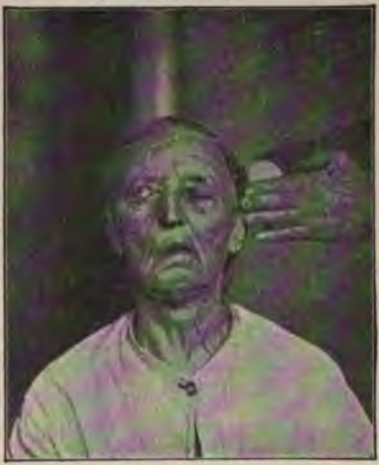
Symptoms.—The paralysis occurs suddenly, in the night, without warning; or it develops slowly, following some other disease. The rheumatic and refrigeratory facial paralysis occasionally has prodromes, especially *pain*, which may last days or one to two weeks before the paralysis appears (Webber, Testaz). This pain is located in the ear or back of it, in the face or on the neck, and indicates an involvement of the sensory nerve-branches, especially of the trigeminal or of those of the occipital and cervical regions. A slight *swelling* of the face, particularly in front of and below the ear, is occasionally noticed at the beginning of the trouble. It is occasionally accompanied by fever, headache, vomiting, tinnitus aurium, etc. The paralysis involves the *facial muscles* supplied by the facial nerve. As a result, an asymmetry of the two halves of the face occurs, which may show itself even when the muscles are at rest, and which is more strongly marked in old people than in the young. The wrinkles of the affected side disappear; the eye is wide open, and to such a degree that the mucous surface of the lower lid looks outward, and the tears do not reach the tear-duct (Fig. 166). The nose inclines towards the sound side; the nasolabial fold is absent on the affected side; and the mouth is drawn towards the other side, while the angle of the mouth stands lower on the paralyzed side than on the other, and the mouth is wide open on this side.

FIG. 165.



Left-sided facial paralysis; involvement of all branches.

FIG. 166.



Chronic facial paralysis (right side). In a woman seventy-five years old. Attempt to close the lids.

The trouble becomes worse on movement: corrugation of the brow, closing of the eyes, sniffling, articulatory and mimic movements, are executed only on the normal side. This makes the asymmetry more evident. The paralysis comes prominently to view on attempts to laugh, to open the mouth, and to show the teeth. The lips cannot be closed on the paralyzed side; the saliva dribbles from the angle of the mouth on that side, sometimes also the food. In doubtful cases the muscular strength may be tested by endeavoring to pull apart the closed lips, or by asking the patient to compress your finger between his lips. The buccinators do not contract; the cheek puffs out on expiration, but does not lie close to the teeth in chewing.

In attempting to close the eye, the upper eyelid is depressed through contraction of the levator palpebræ superioris, and the eyeball turns upward until the cornea is hidden under the lid; but the palpebral fissure, on account of the paralysis of the orbicularis palpebrarum, remains open (lagophthalmos).

When at rest it is generally somewhat wider than that of the other side. The lid-reflexes also are absent, and foreign bodies entering the eye are not removed, and produce a conjunctivitis. If a paresis of the orbicularis palpebrarum is all that is present, the eye may be closed, but the slightest pressure suffices to open it.

The aural muscles and the platysma are not always involved. It is doubtful whether the uvula is ever affected in peripheral facial paralysis (Gowers, Jackson). An oblique position of the uvula denotes nothing, as it occurs also in normal persons. We can only speak of a paresis when the palatal arch is lower on one side than on the other, and when the uvula only slightly contracts during phonation. The articulation is heavy at first, as the labials are hard to form. This, however, soon remedies itself. The defective dilatation of the nostril may cause anosmia.

In many cases the sense of taste is decreased or lost on the anterior two-thirds of the tongue on the same side (involvement of the chorda tympani). Only rarely is sensation decreased in this area (Bernhardt). Total hemiageusia has been observed in a few cases.

We occasionally find a *decrease in salivary secretion* on the paralyzed side (Arnold, Romberg).

The tongue protrudes normally, but the drawing of the mouth which is present sometimes simulates a deviation. Involvement of the stylohyoid and digastric does not produce any marked symptoms, though Schultze has lately described depression of the floor of the tongue as a symptom of facial paralysis, and is inclined to refer this phenomenon to a paresis of these muscles.

Hyperacuity of hearing (oxyokeia) has been observed (Roux, Lucae, and others). It has been referred to paralysis of the stapedius, though Urbantschitsch blames it on an abnormally increased contraction of this muscle.

Herpes zoster and edematous swelling of the paralyzed side of the face (v. Frankl, Hübschmann) are rare symptoms.

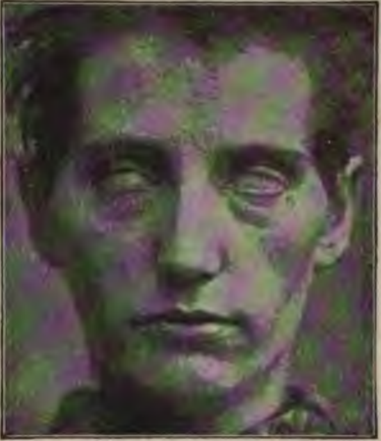
A partial degenerative reaction to electrical stimulation may occur after the first two weeks, but it is not always the case. Sometimes the electrical excitability may be increased.

The symptomatology of facial diplegia does not need a special description; Fig. 167 illustrates it.

Diagnosis. — When completely developed, a diagnosis is not difficult to make.

It is important to locate the lesion. We must distinguish between paralyses produced by a lesion of the cortical centre, or of the fibres which descend from it to the facial nucleus, and those of the nucleus, as also those of peripheral origin. Fig. 168 serves to illustrate these relations.

FIG. 167.



Facial expression in diplegia facialis.

A slight asymmetry of the facial innervation is often found in normal persons. In doubtful cases it is advisable to compare with photographs from some earlier time. The customary contraction of one side of the mouth in smoking, etc., may produce such an asymmetry. Absence of the teeth on one side and atrophy of the alveolar process may also produce an asymmetry and simulate a difference in innervation.

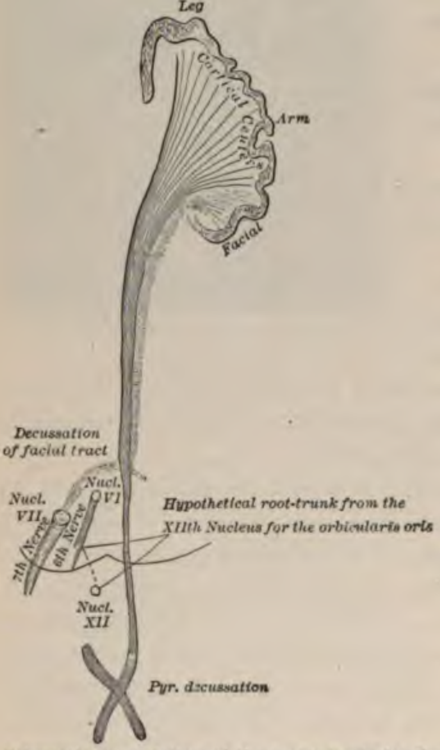
The conducting path for the facial nerve comes from the cerebral cortex, joins with the fibres from the arm and leg centres in the medullary radiations of the inner capsule, and then runs alongside the motor paths for the extremities of the opposite side through the cerebral peduncles to the pons. Here it extends over the middle line to the contralateral side and reaches the *nucleus of the facial nucleus* (Nucl. VII.), which is in the neighborhood of the abducens nucleus.

The peripheral nerve commences at the nucleus, and for a short distance runs alongside the auditory nerve. The path from the cortex to the seventh nucleus in the pons we call the *volitional tract.*

Paralysis of this part produces a facial paralysis characterized by (1) normal electrical excitability; (2) unimpairment of the upper facial; this is possibly due to the existence of a special tract for this part (which I regard as improbable), or perhaps to the fact that the upper facial is innervated from both hemispheres; (3) a paralysis of the extremities generally exists at the same time; only when the tract is involved in its course between its decussation in the pons and the nucleus does a *crossed paralysis* occur; (4) the reflex excitability of the facial region is normal.

Paralysis of the nucleus and the peripheral part of the nerve presents the following characteristics: (1) involvement of all branches—this is the rule, but in pontine affections the upper facial is often spared; (2) *signs of degeneration;* this is lacking in very slight cases only, as the nucleus is the trophic centre for the muscles supplied by the seventh; (3) the *reflexes are abolished.*

FIG. 168.


Leg
Cortical Centers
Arm
Facial
Decussation of facial tract
Nucl. VI
Nucl. VII
7th Nerve
6th Nerve
Hypothetical root-trunk from the XIIth Nucleus for the orbicularis oris
Nucl. XII
Pyr. decussation

Schematic presentation of the motor tracts for the facial nerve and the extremities.

Some peripheral facial paralyses are limited to single branches. This is naturally especially true of some of the traumatic cases. A paralysis of the lower branches often occurs, for instance, after extirpation of the submaxillary and retromaxillary lymphatic glands (Camillo Fürst). Mann and Bernhardt particularly have shown that under other circumstances single muscles, as the orbicularis oris and palpebrarum, may remain uninvolved. An isolated paresis of the orbicularis palpebrarum of doubtful origin has been described by Silex.

A diagnosis between disease of the facial in the pons and a disease of the nerve-trunk may be difficult; but the accompanying symptoms are generally sufficient to clear the view, as the pontine facial is rarely involved without other parts of this region being affected, and the diseases of the facial trunk present many characteristic points which differentiate them from pontine troubles.

It is clear that the pontine lesion is generally accompanied by a paralysis of the abducens. I have, however, seen a case of a peripheral character in which the sixth and seventh were both involved.

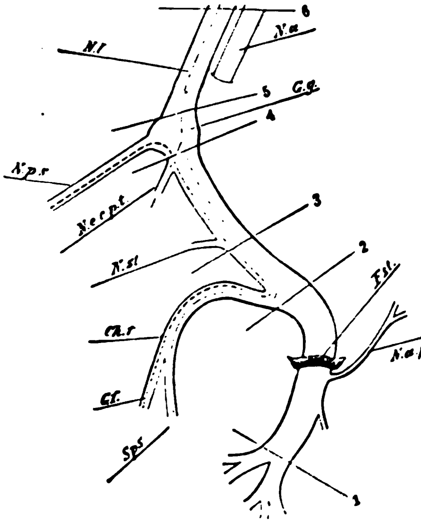
Diseases of the medulla oblongata often involve the labial facial as well as the hypoglossal, giving rise to the view that in the innervation of the obicularis oris the hypoglossal nucleus is also involved. This is indicated in Fig. 168.

If the facial is attacked *at the base of the skull,* we would expect *paralysis of the auditory* and other cranial nerves, together with *general cerebral symptoms* (headache, *vertigo, vomiting,* etc.).

To localize the seat of the lesion in the further course of the nerve, consult Erb's scheme (Fig. 169).

If the lesion occurs between 1 and 2, the facial muscles alone are paralyzed; between 2 and 3, the gustatory fibres of the chorda are involved, and taste is lost on the anterior two-thirds of the tongue. If the disease is between 3 and 4, the stapedius is involved, producing hyperacuity of hearing. If the geniculate ganglion between 4 and 5, or the nerve above this, is involved, the velum on the same side is paralyzed (?). This rests upon the hypothesis that the motor fibres for the palatal muscles leave the facial in the track of the major superior petrosal, and reach the palate through the sphenopalatine ganglion and the descending palatal. We need say no more than that an absolute diagnosis cannot be made from the symptoms or with the use of Erb's scheme, as variations are so frequent and our knowledge of the innervation not too accurate.

FIG. 169.



Showing the facial trunk from the base of the skull to the pes anserinus. *N.f.*, facial nerve; *F.st.*, stylomastoid foramen; *N.a.p.*, posterior auricular nerve; *N.p.s.*, greater sup. petrosal nerve; *N.e.c.p.t.*, communicating nerve. with plex. tymp.; *N.st.*, stapedius nerve; *Ch.t.*, chorda tympani; *N.a.*, acoustic nerve; *G.g.*, gangl. geniculi. (After Erb.)

Course.—Slight cases may recover in one to two or more weeks. Severe cases may last for months or remain stabile. Even if improvement occurs, a new trouble comes on,—*contractures* of the until then paralyzed muscles. The mouth gradually draws to the paralyzed side, the nasolabial folds are furrowed more deeply, the palpebral fissure becomes smaller,—all a result of contraction and shortening of the muscles. The normal side then, on superficial examination, appears to be the seat of the paralysis. Examination of the active movements, speaking, laughing, etc., shows that, notwithstanding the contracture, the active and emotional movements are limited to the side that was formerly paralyzed.

The movements that often accompany this contracture may confuse one. In shutting the eyes not only the orbicularis palpebrarum but also the zygomatic on the side that had been paralyzed contracts, so that the labial angle is drawn to one side. In a physician who had had facial paralysis with ageusia, every attempt to move the paralyzed muscles produced a metallic taste on the corresponding half of the tongue.

These *secondary* phenomena which occur in the course of facial paralysis are supposed to be due to irritation of the nucleus caused by the forcible attempts at innervation or to the secondary nuclear alterations already spoken of. Electrical treatment has also been blamed, but without justification, though it might be imagined that strong stimulation involving the trigeminal could irritate the facial nucleus (as, for instance, a convulsive tic often accompanies a trigeminal neuralgia). They develop, however, also in patients who have not received any electrical treatment.

Recovery from facial paralysis may also be incomplete in that some of the muscles, for instance the muscles of the mouth, again become capable of functionating while the lids can be only partially closed, and *vice versa*.

Sometimes twitchings, the symptoms of *convulsive tic*, are found.

Facial paralysis may remit and intermit, may become partially cured, and may be combined with an hysterical hemianesthesia of the same side (Oppenheim).

The character of the original trouble indicates the **prognosis.** In a rheumatic paralysis the chances for a cure depend upon the severity of the lesion. A facial paralysis which is due to caries of the petrous bone, etc., is incurable.

We may distinguish a *slight*, *severe*, and *medium* form of facial paralysis according to the electrical excitability. It is slight if after two weeks the electrical excitability is normal or only slightly involved, severe when a complete reaction of degeneration is present, and medium when only a partial reaction of degeneration is found. Slight paralyses recover in two or three weeks; medium, in four to six or eight weeks; the severe ones, when at all, in three to six months. Some cases, of course, vary greatly.

In catarrh of the middle ear the prognosis depends upon the cure of this trouble.

Treatment.—The first indication is to combat the original disease. A *specific* treatment for *syphilis*, a *diaphoretic* and *salicylic* treatment for rheumatism, or *bloodletting* or *vesicants* or a *purging* treatment in the first stage of infectious or rheumatic forms, may do good. Hot cloths are recommended by Gowers. If necessary, suturing of the nerve or other procedures may be undertaken. It may be necessary, for instance, to extirpate a tumor, to evacuate an abscess, to treat an otitis media, etc.

Electrotherapy is the most important direct treatment. In fresh cases a stabile galvanic current to the nerves is the best,—the cathode negative electrode (ten square centimetres) on the nerve-trunk, the anode on any indifferent place; slow in- and out-currents of from one to three milliamperes for two or three minutes. In a physician whom I treated in this manner, the taste returned in the affected half of the tongue immediately after the first sitting. It might be advisable to conduct the current directly through the brain, by way of the auriculomastoid fossæ. Later, labile galvanic currents may be used. Strong currents should be avoided, and special caution is necessary in severe cases in using the faradic current. It is advisable to institute electrical treatment as soon as the first signs of contracture appear.

Many persons are so sensitive to electricity that one must begin with very slight currents, and entirely avoid opening and closing of the current. These cases have probably had a neuritis develop in the course of the paralysis. The electrical treatment must be carried on daily at first; afterwards every second day.

I succeeded in improving, through an electrical treatment, cases of facial paralysis which had existed for years. We have no means of combating the secondary contractures. Light massage and stretching of the cheek by means of wooden spheres inserted into the mouth are recommended. Electrization of the muscles of the normal side is of no avail. I have succeeded, in some cases, in improving the condition considerably by ordering exercises in speech and fixing the labial angle of the sound side with gum-plaster or with the hand. It is advisable to cover the eye if lagophthalmos exists.

PRIMARY AND ISOLATED PERIPHERAL DISEASES OF THE AUDITORY NERVE

occur rarely in comparison with the disorders of hearing produced by diseases of the ear. The terminal branches of the nerve in the labyrinth are relatively often involved, perhaps through extension of the disease-process from the middle ear. The nerve-trunk may be injured at the base of the brain by caries, periostitis, tumors of the bones or meninges, aneurisms of the blood-vessels near the auditory nerve, or from an inflammation in the neighborhood of the nerve. A *rheumatic* paralysis of the auditory nerve rarely occurs. Polyneuritis may also involve this nerve (v. Strümpell). A leukemic infiltration of it has been repeatedly observed. It is doubtful whether a *primary inflammation* and *atrophy* of this nerve ever occurs; a calcification or deposit of calcium salts has been observed.

Disease of the auditory nerve in tabes, multiple sclerosis, and other central diseases will not be discussed here.

Diseases of the trunk of the auditory cannot be easily differentiated from labyrinthine diseases. The important symptom in both is *difficulty of hearing*, or deafness, generally combined with *subjective tinnitus*, and often with vertigo and incoördination. This latter is due to involvement of the semicircular canals or of the vestibular nerve.

Inflammations, hemorrhages, and sclerotic processes may develop in both labyrinths at the same time. This could be due to the *acute infectious* fevers (typhoid, scarlatina, malaria, mumps, influenza, etc.) as well as to *syphilis*.

Nephritis, diabetes, pernicious anemia, hemorrhagic pachymeningitis, and leukemia may be the cause of the labyrinthine disease. Diseases of the neighboring organs, and especially *epidemic cerebrospinal meningitis*, may involve the inner ear.

Cases are recorded in which a *hemorrhage* into both labyrinths (after trauma) was the cause of total deafness. The deafness caused by poisons (quinine, sodium salicylate, etc.) seems to be also of labyrinthine origin. Senile involution may also involve the labyrinth and produce a decrease in hearing.

The connection between labyrinthine diseases and vertigo and incoordination will be discussed at length in its proper place.

A basal disease rarely confines itself to the trunk of the auditory nerve; it extends to the facial more often than to the other nerves, and to the medulla, pons, cerebellum, etc.

Concerning the signs of nervous affection of hearing and methods to determine it (Rinne's and Weber's methods, etc.), see page 74.

Subjective tinnitus of the ears is a symptom which can occur in every disease of the auditory system; it is especially persistent in nervous defects of hearing. We may even say that where no subjective tinnitus is present, as a rule no nervous affection is present (v. Frankl).

We must always bear in mind that anemia, congestion, and aneurisms of the cerebral arteries may be the cause of the trouble. Clonic spasm of the inner muscles of the ear is sometimes a cause. This form and that produced by aneurisms may be objectively detected. Another cause of deafness and *tinnitus* is the influence of loud noises—in factory hands, locksmiths, blacksmiths, engineers and firemen on locomotives.

A bilateral high-pitched tinnitus extending to the head has a bad prognosis.

For more complete particulars, consult text-books of ear diseases. We will only lay stress upon the fact that nervous tinnitus may in some cases be relieved by the influence of galvanic currents (anode to the ear of the side affected, opening and closing currents). They should be very weak, and each sitting should last about ten minutes. The iodides and mercury may produce a cure, as it is often a syphilitic disease. Revulsives are also recommended.

PARALYSIS OF THE GLOSSOPHARYNGEAL NERVE.

An isolated paralysis of this nerve is hardly ever observed, and the lack of knowledge concerning it and its functions has not been cleared up by pathology. Even though it is certain that the peripheral ends of this nerve receive and conduct the *sensations of taste* from the posterior tongue and the arch of the palate, some observers believe that these fibres leave the glossopharyngeal to join the trigeminal nerve, while others claim that the chorda fibres pass into the central glossopharyngeal nerve. No case has been observed in which it was absolutely proved that the posterior fibres of taste are found in the trunk or root of the glossopharyngeal nerve.

The case cited by Pope, in which a vertebral aneurism pressed upon the roots of the glossopharyngeal and produced a hemiageusia, is not sufficient proof to me, as the pressure and its results may have involved other organs. The sensory fibres of this nerve reach the pharynx, the tonsils, posterior palatal arch, the tympanic cavity and Eustachian tube, and the posterior part of the tongue. A part of this region is also innervated by the trigeminal. There is no doubt, however, that the sensory pharynx is wholly innervated by the glossopharyngeal. It also takes part in the motor innervation of the pharyngeal muscles, though to what extent is not yet known.

Réthi believes that only the fibres for the stylopharyngeus come from it. According to Kreidl, the motor nerves of the esophagus arise in the glossopharyngeus, but pass to the vagus afterwards.

Ignoring the diseases of the medulla which affect this nerve, and considering only the peripheral diseases, the processes which involve this nerve are inflammations and tumors, especially syphiloma and aneurisms. It may also be involved in thrombosis of the jugular vein, or periphlebitis, or in its extracranial course by injuries, tumors, etc.

A degeneration of this nerve with its roots was observed by me in tabes.

Symptoms of a peripheral affection of the glossopharyngeal nerve are *anesthesia* of the upper half of the pharynx, *ageusia* of the posterior half of the tongue and gums, and *dysphagia* as a result of paralysis of a part of the muscles of the pharynx, also lost reflex excitability of the mucous membrane of the pharynx. These symptoms belong, among others, to the symptomatology of postdiphtheritic paralysis, though ageusia is not found in this affection.

It has been asserted that diseases of the tympanic cavity may produce an ascending neuritis of Jacobson's nerve, which extends to the glossopharyngeal nerve.

PARALYSIS OF THE VAGUS NERVE.

Anatomy and Physiology.—It is very probable that the fasciculus solitarius, the longitudinal fasciculus, forms a common *sensory* root to the vagus and glossopharyngeal, which does not—as was formerly believed—ascend from the spinal cord, but arises in the root-ganglia, enters the medulla oblongata, and passes downward from that region.

We are not positive as to the origin of the vagus (and accessorius). This is especially true of the *motor* root. Most of the late authorities (Meynert, Kölliker, Dees, Grabower, Bunzl-Federn) regard the ambiguous nucleus as the motor nucleus for the vagus. Kölliker includes other cell-groups also. The posterior vagus nucleus is regarded as the sensory terminal nucleus in which the sensory vagus root branches, though lately some authorities have claimed that motor fibres arise in this nucleus (v. Monakow). Marinesco believes that the ambiguous nucleus is the motor nucleus for the striated, the posterior for the unstriated, muscles of the vagus region. Until lately the motor-fibres for the muscles of the larynx which are found in the vagus were supposed to come from the accessory (Bischof, Longet, Bernard, Schiff, and others). Grossmann and Grabower deny this. The latter showed experimentally that the motor fibres for the larynx were present in the lowest four or five root-bundles of the vagus. From the anatomical researches which he carried on in my laboratory, he showed that these fibres come from the ambiguous nucleus, while the accessorius is only a spinal nerve. He claims that there is no such thing as a so-called vago-accessorius. Hall had already expressed the same opinion. Bunzl-Federn, however, was able to follow the accessorius nucleus to the oblongata (in rabbits).

The vagus has an extensive distribution, covering the pharynx, larynx, heart, lungs, esophagus, stomach, and intestines. Through the auricularis vagi, many of its fibres reach the external ear.

It assists in the *innervation of the palate*; according to some it is the only motor nerve of the palate. A branch of this nerve, the *pharyngeal*, forms with the glossopharyngeal (and sympathetic) the pharyngeal plexus, which innervates the pharyngeal muscles and mucous membrane.

The *superior laryngeal* supplies the cricothyroid, perhaps also the thyro-epiglottic and the aryepiglottic, and the mucous membrane of the epiglottis. The *inferior, or recurrent laryngeal*, innervates the rest of the laryngeal muscles and the part of the mucous membrane below the vocal cords.

Section of the vagus in animals produces slowing and deepening of the respiration by interruption of the fibres which reflexly excite the respiratory centres. The superior laryngeal contains centripetally conducting fibres whose excitation makes the respiration slower and deeper, or stops it and closes the rima glottidis. In the branches for the lungs, we have motor fibres for the unstriated muscles of the bronchi; sensory ones for the bronchi and lungs; also fibres which inhibit the inhibitory fibres of the heart,—*i.e.*, which produce a rapid pulse. Bilateral section of the vagus in animals produces death, as, from failure to close the larynx, food gets into the lungs (Traube). Schiff claims that a neuroparalytic hyperemia of the lungs results from paralysis of the vasomotors. Eichhorst regards the vagus nerve as a trophic nerve for the heart-muscles, section of which produces a fattening of these muscles.

The esophageal branches innervate the muscles and mucous membrane of the esophagus. The vagus contains *secretory* fibres for the mucous membrane of the stomach, as well as *vasomotor* ones. It also contains motor fibres for the stomach and, to some extent, for the intestines. It contains both inhibitory and excitatory fibres for the muscles of the heart.

Etiology.—The vagus may be involved by disease processes which develop in its vicinity at various places in its course, but is rarely the seat of a primary neuritis. A rheumatic form of *neuritis*, confined to one or both recurrent laryngeal nerves, has been occasionally noticed. The vagus is often affected in multiple neuritis, this being particularly true of the alcoholic form. Diphtheritic paralysis involves this nerve rather often, neuritic or degenerative alterations of the nerves and their branches being the cause of the paralytic symptoms (R. Meyer, Vincent, and others). Multiple neuritis, typhoid (Lublinski zur Helle), pneumonia (Schroetter, Botkin), scarlatina (Gottstein), malaria (Schech), cholera (Matterstock), influenza (Schmidt, Krackauer, Réthi, Lähr), etc., have been given as causes.

Alcohol, lead, and arsenic (Imbert and Gourbeyre) may poison and paralyze it.

Paralysis of the vocal cords has also been noticed as a symptom of acute atropine and morphine intoxication. Hemorrhage into the vagus trunk was observed by Richel in phosporous poisoning. The vagus symptoms of tabes are mostly of bulbar origin, but may be due to a degeneration of the nerve, as I have shown.

The vagus symptoms occurring in diseases of the medulla (tumors, softening, hemorrhages, bulbar paralysis, tabes, multiple sclerosis, etc.) are discussed elsewhere.

Meningitic exudates, hemorrhages, tumors, aneurisms, periostitic and carious products at the base of the skull may injure it in its *intracranial* course. Arteriosclerosis, especially of the vertebral and inferior cerebellar arteries, thrombosis of the transverse sinus and jugular vein (Stacke and Kretschmann, Schwarze, Kessel), suppuration (Martius), tumors of the neck or mediastinum, mitral stenosis from dilatation of the auricle (Ortner), may all involve this nerve. It may also be affected by wounds or operations on the neck (ligation of the carotid, extirpation of tumors), by tuberculosis (pleuritic nodes or enlarged lymph-glands compressing it), by pericarditis, and by neuroma.

The vagus symptoms occurring in the course of the functional neuroses (hysteria), as well as in anemia, are of central origin, and are spoken of elsewhere.

Symptoms.—To a certain extent the symptoms are dependent upon the location of the lesion. Signs of a complete vagus paralysis are observed particularly in processes which involve this nerve at the base of the brain. In such cases other cranial nerves are almost always affected at the same time, especially the upper roots of the eleventh and generally the ninth and the twelfth. If only one side is involved, we have *unilateral paralysis of the palate, the larynx, and the fauces.* The velum palatinum hangs loosely, and does not move in phonation; the speech is nasal; dysphagia is very slight. Erben found the posterior wall of the pharynx excavated, the hyoid bone and the larynx pushed to the normal side. This does not always occur (Möbius). The vocal cords remain in the median line, but do not take part in phonation or in respiration. Electrical exploration has occasionally revealed the degenerative character of this paralysis. Anesthesia of the pharynx and larynx has but rarely been noticed. The same symptoms, with the exception of the paralysis of the tongue, occur in injuries of the vagus high up in the neck. Deglutitory paralysis in extracranial diseases of the vagus is, however, rarely marked.

The heart symptoms are inconstant in unilateral affections; at times retardation, but more often acceleration, of the heart's action is noticed; for example, in compression through tumors (Hayem, Riegel, Stix, and others). In unilateral vagotomy all symptoms may be missing (Weidner, Gurfein).

Respiratory disorders occur, as far as they are not of laryngeal origin, particularly in central diseases and bilateral lesions of this nerve. Sometimes retardation, on other occasions acceleration and irregularity of the respiration were noticed. Continued disturbance of respiration was not observed in any case of unilateral section of the nerve (Traumann).

Other symptoms of irritation and paralysis which are referred to the vagus, which, however, occur rarely in peripheral diseases of the nerve, are vomiting, bulimia, absence of any feeling of hunger and thirst, pain in the stomach, and diabetes (Henrat).

Deglutition or aspiration pneumonia may be a result of section of the vagus. Symptoms indicating a paralysis of the vasomotor nerves of the lungs were absent in this condition.

The most important component of this set of symptoms is the laryngeal paralysis, which may also be observed alone, and as often from disease of the vagus itself as from disease of the inferior or recurrent laryngeal.

A complete description of these paralyses would bring us into the domain of laryngology; hence they will be but lightly touched upon.

Paralysis of the recurrens nerve presents the following symptoms. The vocal cord of the same side remains in the cadaveric position (midway between adduction and abduction), and is immovable during phonation as well as during respiration. The researches of Grossmann show that the vocal cords may first assume a median position (action of the *cricothyroid?*), and later assume a cadaveric position. The voice is always raw and hoarse, even though there are no other changes noticed. Deep inspiration may be accompanied by stridor.

In bilateral paralysis of the recurrent laryngeal we have aphonia, the glottis not closing even in coughing, inspiratory stridor, and dyspnea.

A bilateral paralysis of the phonators is perhaps always of central origin, and more often a symptom of hysteria than of organic diseases of the oblongata. The organic diseases produce only respiratory paralysis, or disorders of respiration and phonation together. A partial paralysis of the muscles of phonation is, however, occasionally observed in organic diseases of the nervous system, as multiple sclerosis, bulbar paralysis, etc.

Lead poisoning may produce a bilateral paresis of the internal thyro-arytenoid muscles, also paralysis of the posterior crico-arytenoid.

Paroxysmal tachycardia, asthma nervosum, etc., will be discussed elsewhere.

Treatment.—In syphilitic cases give potassium iodide and mercury. In alcoholic paralyses give large quantities of alcohol (wine, cognac) and other excitants. These are contra-indicated in other forms.

In saturnine laryngeal paralyses order baths, purgatives, and potassium iodide. Lymphoma may occasionally undergo resorption through the action of iron iodide, arsenic, and a strengthening diet.

Electrical treatment is of doubtful advantage in laryngeal paralysis of an organic nature. Not a few cases of rheumatic paralysis of the recurrens have, however, been described in which faradization is said to have brought about a prompt recovery. It is advisable always to apply it externally, and never directly to the larynx. Intralaryngeal stimulation may in paralysis of the posticus increase the trouble, in that it stimulates the tensors of the vocal cords, both directly and reflexly. In order to stimulate the recurrent laryngeal externally, place a knob-like electrode (cathode) between the inner border of the sternocleidomastoid and the larynx at the height of the cricoid cartilage and press it deeply inward and downward. It is then possible with a strong galvanic current to stimulate the sound nerves with good results.

The larynx may be massaged in the following manner: Pressure on the posterior part of the thyroid cartilage produces a closing of the vocal cords, the arytenoid cartilages approaching each other when the patient is asked to phonate. Hysteric paralyses of phonation are especially amenable to this form of treatment. Posticus paralysis may demand the performance of tracheotomy.

PARALYSIS OF THE ACCESSORIUS NERVE.

In accordance with what we have written in the previous chapter, we cannot speak of an inner branch of the accessorius, the vago-accessorius, as this fasciculus is a part of the vagus from its very origin. But some are inclined to believe that the accessorius, in addition to its spinal nucleus, which arises in the external ramus, arises also from cell-groups in the oblongata.

The external ramus supplies the *sternocleidomastoid* and the *trapezius*. Some few fibres from the second and third cervical nerves pass to the sternomastoid, but it is almost entirely under the control of the accessorius. The cervical nerves take part in the innervation of the trapezius, so that a paralysis of the accessorius does not always paralyze the trapezius. This is particularly true of the clavicular portion of the muscle. The cervical nerves exert only a slight influence upon the innervation of the middle portion. This explains why the observations of different authors (Remak, Bernhardt, Schmidt, Schlodtmann, etc.) concerning the condition of the middle trapezius bundle in paralysis of this nerve do not harmonize. In a case examined by me, in which the accessorius on both sides was cut and a large piece excised, for the cure of spasms in this region, the sternocleidomastoid was completely inert, while the trapezius, though weak and atrophied, still had some fibres which were capable of being contracted. An "angel-wing" position was merely indicated.

Myelitis cervicalis superior, progressive muscular atrophy of spinal origin, gliosis cervicalis, and other diseases of the cervical cord, may injure the nuclei and roots of the accessorius. In addition, *caries* of the cervical vertebræ, with *compression* of the nerve-roots from tubercular granulations and abscesses, *peripachymeningitis tuberculosa and syphilitica, neoplasms and meningitic* exudates in the region of the foramen magnum, also injuries of the nerve itself and compressions from tumors, may all be causes.

A primary *neuritis* of the nerve may occur. It is uncertain whether the accessorius paralysis noticed sometimes in tabes is of central or peripheral origin. The accessorius paralysis may be unilateral or bilateral, generally the former.

The symptoms are motor ones: paralysis of the sternomastoid and partial, rarely complete, paralysis of the trapezius. The first need not produce a deformity; this only occurs when a secondary paralysis develops in the muscle of the other side. Paralysis of the sternocleidomastoid is shown by the fact that the head or chin cannot be moved much to the opposite side, and that in attempting to do this the sternocleidomastoid does not contract. Its belly does not bulge out in inspiration either. In bilateral paralysis of the sternocleidomastoid the head falls easily backward and cannot be easily inclined.

The anomalies of position produced by complete paralysis of the trapezius have been already described on page 25. We wish merely to add here that partial paralyses which are often noticed do not present the *"angel-wing" position of the shoulder-blades* which should occur. The paralysis—except in the milder cases—is a degenerative one, and characterized by the well-known alterations in electrical excitability.

Prognosis and treatment depend on the cause. Syphilitic cases are especially amenable. Electricity must be used to combat the paralysis. Gaupp has recommended a support which draws back the sunken shoulders and allows the other muscles to develop.

E. Remak has called attention to the fact that the symptoms arising from section of the accessorius are less marked when it occurs high up in the neck than when it occurs near the entrance of the nerve into the trapezius, as here the cervical branches have already joined the nerve.

PARALYSIS OF THE HYPOGLOSSAL NERVE.

The twelfth nerve is more often injured in its intracerebral and bulbar than in its peripheral course. Of seventy-nine cases which Ascoli collected from the literature on the subject only one-third were of peripheral character. Hypoglossal paralysis is almost always a symptom of hemiplegia, because the intercerebral tract, which descends from the cortical centres to the nucleus for the nerve in the medulla, joins the tract for the extremities. Atrophy of the tongue, however, never accompanies it, because the trophic centre is in the hypoglossal nucleus in the medulla oblongata.

Diseases of the *medulla oblongata* generally produce bilateral hypoglossal paralysis and atrophy of the tongue, which is almost always combined with paralysis of other cranial nerves. A *unilateral nuclear disease* has, however, been observed.

As causes for the *peripheral* hypoglossal paralysis, we have disease-processes in the posterior cranial fossa,—tumors at the base of the skull, meningitic exudates, basal hemorrhages, carious processes and also *aneurisms of the vertebral artery, luxations of the upper cervical vertebræ,* and hydatid cysts (Dupuytren). The hypoglossal nerve may be injured in the neck by different *traumata*, compressed by *tumors*, or injured in the extirpation of tumors. Cases of this kind have been described by Hutchinson, Weir Mitchell, Schüller, Remak, Traumann, Paget, and others.

A simple isolated neuritis may occur, though very rarely (Erb, Montesana, Marina). In some of the cases described as such, however, an acute infectious disease preceded it (scarlatina, angina).

It is doubtful whether hemiatrophia linguæ can be of *toxic* origin (lead, arsenic, alcohol).

Hemiatrophia linguæ may, however, be present in tabes, syringomyelia, multiple sclerosis, as a congenital symptom, alone or in conjunction with atrophy of other cranial nerves or with muscular defects, and as a component of hemiatrophia facialis.

Peripheral hypoglossal paralysis produces paralysis and atrophy of the corresponding side of the tongue. When lying on the floor of the mouth, the tongue does not deviate or its tip is inclined towards the normal side. Traumann believes this to be due to the fact that the tonicity of the longitudinal muscles shortens the normal half of the tongue. Gowers claims that the root of the tongue is higher on the paralyzed side than on the sound side, due to the loss of tonic contractions of the posterior fibres of the hypoglossal muscle; I have noticed the same. It is difficult for the patient to move his tongue inside his mouth towards the paralyzed side, but this inability is never marked. When extended it deviates to the paralyzed side, and the raphe forms an arch whose concavity is turned towards the paralyzed side. (Fig. 35.) This deviation is due to the atrophy of the genioglossus, whose contraction draws it to the opposite side. This deviation of the extended tongue may be absent in cases of partial paralysis.

Atrophy is also present; the tongue is wrinkled on the diseased side, is contracted, is not as broad, feels soft and flaccid, and has a fibrillary tremor. Electrical examination reveals an incomplete reaction of degeneration. An involvement of the external laryngeal muscles (sternohyoid, sternothyroid, and omohyoid) has rarely been observed (Möbius, Remak).

The motor fibres for these arise for the most part in the upper cervical roots, so that lesions above the place where these fibres join the nerve do not produce any paralysis of them. In unilateral paralysis of the tongue, we do not have very much disturbance in function. The speech is somewhat involved, and the tongue may be slightly hindered in its movements. The speech disturbance is slight even in complete hemiglossoplegia, but may be considerable even in partial bilateral glossal paralysis. The impairment of deglutition and mastication appears only in bilateral hypoglossal paralysis, which is, however, rarely of peripheral origin. Recovery has been noted several times in syphilitic, traumatic, and rheumatic cases. I saw a case of lingual hemiatrophy following upon vertebral caries recover.

The treatment is similar to what was indicated in discussing the therapy of the diseases of the other cranial nerves.

MULTIPLE NEURITIS (POLYNEURITIS).

The causes of this disease—for our knowledge of which we must particularly thank Leyden, although Duménil, Lancereaux, Leudet, and Eichhorst, described cases before him—are numerous.

Toxic causes are the most common. Alcoholism is especially prone to produce neuritis, not alone the whiskey-drinker, but also the beer-drinker, being affected; but less so the imbiber of wine. *Lead, arsenic, copper, mercury, carbon dioxide, anilin,* ptomaine, and leucomaine poisoning, etc., are factors. As a second class of causes we include the *infectious fevers* (*typhoid, variola, scarlatina, influenza*), *erysipelas, pneumonia,* purulent pleuritis, *acute articular rheumatism, parotitis,* gonorrhea, dysentery, and *diphtheria.*

We have also a *septicemic* and a *puerperal* form. Auto-intoxications, as from gastro-enteritis (Wagner), obstipation, and putrid bronchitis (Minkowski), have been given as causes. Icterus and hepatic cirrhosis have also been cited as causes.

Tuberculosis, syphilis, malaria, and diabetes mellitus are all etiologic factors.

Those cases previously called *spontaneous,* and which in development and course correspond to the type of an infectious fever, are probably of *infectious* origin.

The hypothesis has been set up, that the microparasites of pneumonia, of acute articular rheumatism, of cerebrospinal meningitis, etc., may, under certain conditions, directly excite a polyneuritis, and not first the infectious disease.

Beriberi (kakke) is probably an endemic form of this disease.

Eisenlohr in Germany, and Hammond in America, have both described endemics of multiple neuritis.

Severe types of *anemia, cachexia,* and *senility* may produce degenerative processes in the peripheral nervous system which resemble multiple neuritis. Those occurring in the aged are probably to some extent due to arteriosclerosis. Many have thought that they traced the cause to vascular diseases—arteriosclerosis and arteritis obliterans—(Oppenheim and Siemerling, Joffroy and Achard, Gombault, Lorenz, Schlesinger, and others).

Exposure to *cold* is also given as a cause. It is not possible, however, that it can produce the disease unless some other condition has already acted upon the nervous system. It is probably an exciting cause. I have seen many cases in alcoholic subjects commence immediately after a severe cold.

Combinations of the above factors are especially liable to produce multiple neuritis, as combined alcohol and lead poisoning, *combined action of alcohol* and *some infectious disease* (pneumonia, tuberculosis), etc. The causal connection is not always clear, as those attacked by multiple neuritis are very sensitive to other infectious diseases. I have observed angina, diphtheria, pneumonia, tuberculosis, typhoid, etc., occur in the course of a multiple neuritis.

Persons of from twenty-five to fifty years of age are the most often affected by this disease. It rarely occurs in children, if we exclude the diphtheritic form, nor is it often found in persons of old age except in the forms of cachectic and senile multiple neuritis. *Hereditary and neuropathic diatheses* do not play much of a part in the causation of this disease. There is a familial type of multiple neuritis—progressive interstitial hypertrophic neuritis of childhood (Dejerine, Sottas), which, however, deserves especial notice. (Page 179.)

Symptomatology.—We will take the alcoholic form as a type and follow its description by the distinguishing characteristics of the other forms.

Alcoholic neuritis or alcoholic paralysis develops *acutely* or *subacutely*. It may accompany delirium tremens or result from it. Occasionally alcoholism is the predisposing cause, while a cold or a fever forms the exciting cause. The temperature may be elevated at the commencement or in its further course; but an apyrexial course is not uncommon.

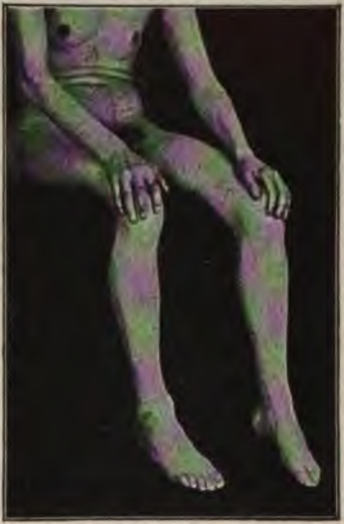
The patients notice first *paresthesia* and *pains*. They complain of a feeling of formication or numbness in the feet and finger-tips, or dull and irritating pains—rarely lancinating—in the extremities, especially the legs. These pains are often slight, but may be severe, and are increased by movements, by pressure on the nerves and muscles, and occasionally by mere touch.

The legs soon begin to feel *weak*, till—within a few days or weeks, perhaps longer—the patient cannot walk. Let us examine him in this stage.

His sensorium is normal, or he is delirious. In addition to the signs of a chronic alcoholism, the gastritis and tremor being especially prominent, we find the following:

We notice a slight *emaciation* of the legs, which is absent in the first stage, or which may be masked by edema and later by fatty deposits. We find the feet inclined downward (foot-drop). The muscles feel flaccid and soft. Pressure upon them produces pain; the nerves, especially the peroneal, tibialis posticus, and crural, are also sensitive to touch. Occasionally we can detect by palpation some swelling of the nerves. Passive movements are uninvolved, but on account of the pain may be limited in extent.

FIG. 170.



Bilateral peroneal paralysis in alcoholic neuritis.

The deep reflexes are annulled, or even with reinforcement produce only slight and uncertain reflex actions. Sometimes, however, at the beginning of the disease they may be increased. The paralysis is a partial one. The *peroneal* nerves are the first that are involved (Fig. 170), occasionally also the *tibialis posticus*, and often also the crural nerve. The paralysis embraces all the muscles innervated by a certain nerve, or spares some, as, for instance, the tibialis anticus in paralysis of the peroneal nerves. In severe cases, and at the height of the disease, all the nerves may be involved, and a more or less complete *paraplegia* be present. Its *peripheral* character is, however, recognized from the fact that some nerve-zones are more involved than others.

Another characteristic of this paralysis is its *degenerative* character; muscular degeneration always occurs and the alterations of electrical excitability are rarely absent. We find a complete or partial *reaction of degeneration*, generally the latter; also faradic reaction of degeneration, and, in certain regions, a simple quantitative decrease of excitability. These phenomena of degeneration may even be found in nerves which are not paralyzed. The upper extremities are either not involved or only slightly. It is uncommon for the disease to start in the upper extremities. The extension of the paralysis to the arms in cases where a diffuse paresis (or paraplegia) exists in the lower limbs, may be of great diagnostic importance. Great variation exists in the area involved. The neuritis may confine itself to one extremity or to a few nerves of the limb, or it may attack a few nerves in the different extremities, or it may involve almost the whole peripheral nervous apparatus.

The *musculospiral* is the most often affected of the brachial nerves, although other nerves may be involved at the same time or singly. We find here also that a part of the muscles under the control of a nerve may be paralyzed while the others are capable of executing their movements.

The *supinators*, for example, sometimes also the abductor longus pollicis may be spared, while the rest of the muscles innervated by the musculospiral are completely paralyzed. It may, perhaps, at the onset involve only the extensor communis digitorum. The *distal parts* of the legs and arms, *the muscles which move the feet and hands*, are the ones, then, that are first, or the only ones, involved.

This degenerative paralysis, which is almost always a *symmetrical* one, presents such a characteristic picture that a diagnosis can often be made with some degree of accuracy at first glance.

If the patient can walk, his gait shows many peculiarities produced by the bilateral peroneal paralysis. It is also made uncertain and labored by reason of the weakness in other muscular regions.

Occasionally *ataxia* is present, which in some cases may even be the most prominent symptom. Through it the active movements and gait are considerably modified. It has even been attempted to separate the ataxic from the motor form (peripheral neurotabes of Dejerine). These cases of incoördination without paralysis are, however, rare. The ataxia rarely involves the arms. When ataxia is present, *spontaneous twitchings*, of which the patient is entirely oblivious, are occasionally noticed by other people.

The *sensory* disorders are less marked than the motor weakness. They are, however, rarely completely absent. They extend over more of the peripheral region than does the paralysis. We find on the soles of the feet decreased sensation for all qualities, and often we find a very peculiar and almost pathognomonic *combination of anesthesia and hyperesthesia*, especially an anesthesia to touch and a hyperalgesia. Or there may be decreased sensation to touch and decreased muscular sense with hyperalgesia. Or we may find hypalgesia with increased sensation for touch.

This hyperesthesia is found particularly in the *planta pedis*, and may be a hinderance to walking. A decrease of muscular sense in the toes is at times the only objective sign of sensory disorder. It has been noticed, too, that hyperesthesia may be present in one section of the skin while hypesthesia is found in another. Delayed sensations, etc., are also often found. The sensory trouble is less marked in the hands; a slight decrease of sensation on the tips of the fingers may be the only indication that the nerves of the upper extremity are involved.

The *skin reflexes* are either annulled or decreased. When hyperesthesia is present, they may be increased in the muscles which are not paralyzed. The ataxia and anesthesia in a few cases form the principal symptoms. In one case, for instance, I found a weakness of the extensor longus hallucis with reaction of degeneration, though at first it seemed as if the sensory disorders and ataxia were the only symptoms present. It is, however, to be noted that the so-called "acute ataxia" (see page 207) may be produced by multiple neuritis.

Vasomotor, *secretory*, and *trophic* disorders are occasionally noticed. The most common is *hyperidrosis* of the hands and feet. *Edema* occurs rather often. It develops in the distal part of the extremities; occasionally, also, in the affected nerves and muscles. In a few cases *swelling* of the joints occurred, which was produced by an exudate of fluid. It may simulate acute articular rheumatism. The skin of the feet is often *reddened*, and feels warmer than normal. Skin eruptions, ulcers, etc., occur but rarely.

The *bladder* and *rectum* remain normal, thus furnishing an important point for differential diagnosis between multiple neuritis and spinal-cord diseases, which are symptomatically allied. Unfortunately, exceptions sometimes occur: weakness of the bladder or strangury may be present. If incontinence of urine or of the bowels occurs with the delirium or in the condition of psychic confusion or stupor which sometimes accompanies the disease, it should not, as a result of this mental disturbance, be regarded as of much pathognomonic importance. If, however, this symptom is found present, with a free sensorium, and continues, it is probable that the spinal cord is involved. The same is true of impotence. Amenorrhea may develop in the same manner (Buzzard).

A girdle-pain (cuirass) is occasionally found, though it does not belong to the symptoms of multiple neuritis.

The functions of the *brain* and cranial nerves are often impaired.

The *psychic* disturbances which occur in the course of an alcoholic neuritis ("polyneuritic psychosis," Korsakow) consist especially in a condition of *confusion and weakness of memory*, so that the times of events are lost and things of the past placed in the future, etc. A deceptive and deceitful memory, illusions, and hallucinations may be present. The patient may, for instance, although he has been lying in bed for weeks, claim that he was riding a few days ago, that he met a few friends at a certain place, and that he not long before saw some of his dead relatives. These conceptions are, however, not lasting, may be easily repressed, and change rapidly in nature. This psychosis is thought to be analogous to the amentia of Meynert. Charcot denied the existence of a polyneuritic psychosis, though psychic disturbances of the above nature have been described in polyneuritis following typhoid, influenza, and in the puerperium (Blocq and Marinesco, Köhler, Collatz, Redlich). Gudden claims that the psychosis is not a part of the polyneuritis, but may occur without it.

Of the cranial nerves, the *ocular nerves* are the ones most often involved. Paralysis of the *abducens, oculomotor*, or some of its branches, may occur; but reflex pupillary rigidity is seldom seen. In many cases nystagmus is present, as I have shown. The optic nerve is rarely involved, though *neuritis* and also *partial atrophy* (especially pallor of the temporal side) are found at times. Uhthoff has seen many cases of this nature, and has been enabled to study the anatomic alterations. The disturbance of sight is generally of the character of a *central scotoma* (especially for colors); this is not only true for optic disease which occurs in combination with alcoholism, but has also been seen in a case of multiple neuritis of *carcinomatous* origin.

Diplegia facialis has been found by myself and others.

The paralysis of the vagus and phrenic, which sometimes occurs, is of much importance. The vagus disorder produces acceleration of the pulse (rarely retardation); that of the phrenic, paresis or paralysis of the diaphragm. Sensitiveness of these nerves, and, in the phrenic, occasionally a loss of electrical excitability, are noticed. Masticatory and deglutitory disorders are rare.

Paralysis of the vocal cords is rarely noticed. In a few cases I was able at the height of the disease to detect a systolic murmur of the heart, with dilatation of it. V. Strümpell cites a case with involvement of the auditory nerve.

It is probable, but not positively known, that a polyneuritis limited to the cranial nerves—a multiple cranial nerve paralysis of neuritic origin—really occurs (Hösslin, Mannaberg).

Concerning the **course and prognosis** the following facts are noteworthy. Alcoholic neuritis generally runs an acute or subacute course. It reaches its acme in a few weeks or months, remains stationary about the same length of time or longer, and then recedes, the nerves last involved recovering first. Cases of a violent character with high fever have a very bad prognosis. They may end in death in from eight to fourteen days, occasionally simulating *Landry's paralysis* (see page 332). Eichhorst speaks of a "neuritis acutissima progressiva." An apoplectic onset has been observed in a few cases (Dubois, Dejerine). If the development is a little less rapid, the prognosis will depend in general upon the constitutional condition and upon the intensity and degree of the paralysis, as well as upon the involvement of the cranial nerves. If marked marasmus or a condition of exhaustion from an infectious disease is present, life is always in danger. Involvement of the vagus and phrenic nerve clouds the prognosis, though even in such cases a favorable ending is possible, as I have observed in some cases. If the legs alone are involved, there is a better chance for recovery than if the paralysis had extended to the arms, and particularly to the trunkal muscles. A limitation of the paralysis to the distal portions of the limbs gives a better chance for recovery than if the paralysis had involved the whole extremities. Only rarely is the disease chronic, and then it runs a chronic progressive or a step-like course. There is also a form of polyneuritis which remits. A few cases of this type have been described by Sherwood, Eichhorst, Sorgo, myself, and others.

In the majority of cases the course is favorable. Complete recovery, or in some cases recovery with a defect, occurs. Convalescence, however, is very slow. The symptoms of irritation generally disappear first,—*i.e.* the pains and the pain on pressure. The hyperesthesia, however, exists for a long time, and during convalescence, at the first efforts at walking, may be very disturbing. I have seen patients in whom the symptoms of paralysis had completely disappeared, and who, nevertheless, on account of the hyperesthesia of the soles of the feet, were prevented from walking for months. Until all the symptoms of paralysis have disappeared, one or two years may have elapsed. In some cases a number of the affected muscles are chronically affected, such residual paralyses being observed especially in the peroneal region. Contractures in the antagonists of the paralyzed muscles, particularly in the calf muscles, sometimes occur; these cases are very difficult to treat. Unfortunately, remissions, which supervene not only through renewed action of the same causes, but also from other noxious agencies (as, for example, cold, over-exertion, falls, etc.), are not rare.

LEAD PARALYSIS.

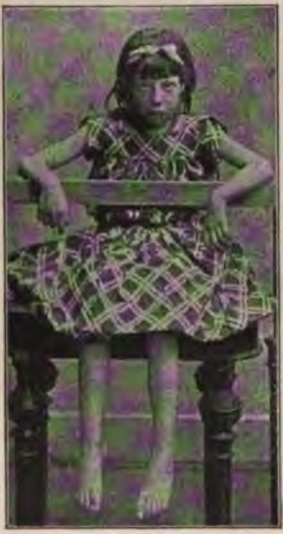
This is a peculiar form of neuritis which generally confines itself to a certain area, and seldom involves the sensory fibres.

The cause is chronic lead poisoning, which occurs especially in workers in lead works, in type-setters, in varnishers, painters, pipe-layers, etc. Formerly the workers in potteries, on account of the preparation of glazes containing lead, and weavers, from working with lead weights, were subject to this form of intoxication. Poisoning from this cause occurs more rarely from drinking water out of lead pipes, through the use of cosmetics which contain lead, and from the washing out of bottles with lead shot. I have also seen it occur in persons whose work consists in sealing cans in canning factories. As a rule, other symptoms of lead poisoning precede the onset of the paralysis, particularly *lead colic*, and occasionally articular and muscular pains (arthralgias) or symptoms of cerebral disease, due to lead intoxication (encephalopathia saturnina). We often find in these persons the so-called *lead-gum,—i.e.,* a blue-black coloration of the gums close to the teeth. Anemia is generally present, occasionally cachexia, arteriosclerosis, and nephritis. The paralysis can, however, be the first and the only sign of the poisoning.

It is noteworthy that lead intoxication may produce disease in the descendants. The children of lead-workers often die from epilepsy (Berger). Other neuroses and organic diseases of the central nervous system (Legrand, Roques) have been observed. I have seen a case of lead paralysis which could only have occurred through hereditary influences (Fig. 171). It affected the radial and the peroneal regions in a typical manner. The lead paralysis in children seems to involve the lower extremities more than the upper (Putnam and Newmark).

The disease generally involves both upper extremities at the same time (the one most used may be involved the most); it is, however, not uncommon for it to be confined to one arm. It develops in the course of some weeks, more rarely in an acute manner soon after an attack of colic. The *extensors* of the hand and fingers are generally, and in many cases exclusively, the muscles affected. If the paralysis is very slight or recent, it may be limited to the extensor cummunis digitorum or to a few of its branches, so that, for example, extension of the third and fourth fingers is impossible. In completely developed cases, however, it is not only the extensor communis digitorum which is involved, but also the extensores carpi, though the extensor carpi ulnaris as well as the extensors and finally also the long abductor of the thumb may be spared. The hands and fingers are found in a position of flexion, and if removed from this position they immediately return to it. The position itself is somewhat characteristic (Fig. 172). As a result of it, the strength which can be exerted to flex the fingers or to shake hands is markedly lowered, but reaches a normal degree when the hand is passively brought to a position of extension.

FIG. 171.

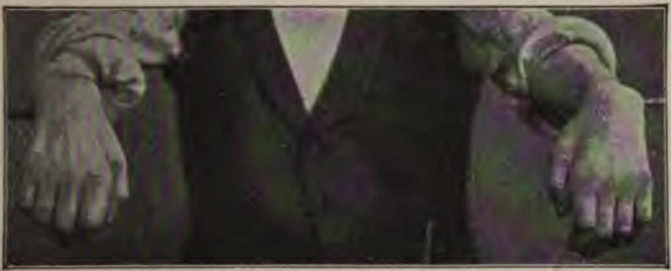


A child with paralysis of the extensors of the hands and feet (probably hereditary lead paralysis).

It can be regarded as a law that in typical cases of lead paralysis the supinators, particularly the supinator longus, as well as the triceps, are spared. It is, however, not so rare to find that the disease has not limited itself to the radial nerve, but has extended to the small muscles of the hand and of the thumb supplied by the median and ulnar nerves, paralysis of these tending even to precede that of the abductor pollicis longus and occasionally involving also the interossei, though the latter are seldom completely paralyzed. It is also not rare to find the deltoid muscle involved, in addition to the extensors.

Atypical cases of lead paralysis also occur in which the supinators are involved together with the muscles of the upper arm,—the biceps, the brachialis internus, and the deltoid (Remak's upper-arm type). The localization may be influenced by the manner or the kind of work done, or by over-exertion of a certain muscle group (toxico-professional paralysis).

FIG. 172.



Position of the hands (wrist-drop) in a case of lead paralysis. The interossei were affected, as well as the extensors of the hands and fingers.

Saturnine paralysis is always a *degenerative* paralysis. The degeneration of the muscular tissue is evident in the wasting of the muscles. It can always be recognized from the reaction of degeneration, which is occasionally observed in muscles which are not involved in the paralysis. Alterations in electrical excitability are distinctly noticeable eight days after the onset of the paralysis. Fibrillary tremor is generally present; sometimes a saturnine tremor.

Swelling over the wrist-joint occasionally is present, due to thickening of the tendon sheaths of the long extensors of the fingers. Thickening of the metacarpal bones is more rarely found.

Sensibility is not disturbed; paresthesia and pain are also generally absent.

In atypical cases in which the paralysis developed in regions which had been overworked, I found sensory disturbances,—for example, in the axillary region in a lead-worker, who had also been engaged in carrying heavy sacks, and in the ulnar region in a tinker, etc. Such cases are, however, rare.

The lower extremities are not often involved. The extensor cruris and the iliopsoas may be temporarily paralyzed. The typical lead paralysis of the legs is, however, that of the peroneal nerve without involvement of the tibialis anticus muscle. There is also a generalized saturnine paralysis beginning acutely with fever in the muscles of the arms, legs, and trunk, following a saturnine encephalopathy.

A diagnosis is easily established in typical cases, as such an extension of the paralytic symptoms very rarely occurs in neuritis from other sources, and is seldom observed in poliomyelitis. In atypical cases the diagnosis can be established from the anamnesis as well as from objective signs of lead intoxication (lead-gum, saturnine tremor, etc.).

It should be noticed that lead intoxication occasionally causes paralysis in the cranial nerves,—for example, paralysis of the laryngeal muscles, paralysis of the vocal cords, and paresis of the adductors and of the abductors. The vagus may also be involved, causing rapidity of the pulse and disturbances of respiration. The optic nerves may be independently involved (amblyopia without any anatomic lesion, optic neuritis or atrophy), or be affected in the course of a saturnine encephalopathy.

To such causes have been ascribed the different cerebral symptoms and cerebral symptom-complexes which follow lead intoxication. These include true *apoplexy* (hemiplegia and aphasia), which follows hemorrhages and, more rarely, softening, and also *transient focal symptoms* whose anatomic basis is not known,—for example, *amaurosis*, *hemianopsia*, etc., and particularly *delirium*, *coma*, and *convulsions*. Though these disturbances are generally acute and disappear either rapidly or end in death (death usually occurring in coma or during convulsions), there are others which develop in a chronic manner, or after an acute onset take a chronic course. To these belong hysteric phenomena, particularly *hemianesthesia* with corresponding sensory disturbances, spasms of an hystero-epileptic character, hemiparesis of a functional paralytic character, etc. Epilepsy and the various psychoses—particularly a curable form similar to paralytic dementia, and probably also a true form of this mental disease—must in some cases be regarded as a product of chronic lead poisoning. Lead may also act directly upon the brain, and in this manner produce general cerebral symptoms and focal phenomena, particularly those peculiar neuroses allied to hysteria. At the same time some of the cranial nerves—for instance, the optic—may be anatomically involved. It can also act upon the cerebral vessels and produce an *arteritis*, which causes *hemorrhages* and *encephalomalacia*, and lastly can evoke *nephritis* with uremic cerebral symptoms. All these factors may occur at the same time. A. Westphal has described this in full in his dissertation written under my control.

The prognosis as to life is good, providing grave complications, as saturnine cachexia, nephritis, etc., do not occur, and is also good as to the curability in the first stage, providing the paralysis is not extensive and has not existed too long. Even a reaction of degeneration does not justify a gloomy prognosis, as recovery is the rule if conditions favor protection of the individual from the poison. Repeated attacks of paralysis make the prognosis worse. The remissions are not only caused by a new intoxication, but also by other injurious agents, as, for instance, over-exertion of the muscles.

ARSENICAL PARALYSIS.

This is a rarer form of multiple neuritis than those previously described, though it occurs often enough to prove that caution should be used in administering this drug medicinally. The paralysis generally follows an acute poisoning, as, for instance, in many cases where arsenic or Paris green has been taken with suicidal intent. But it also occurs after subacute poisoning, as in the medicinal use of the drug in chorea and other diseases (observations of Brouardel, Barrs, Raymond, and others), and it also develops after chronic poisoning.

Gastro-intestinal disturbances follow the symptoms of nervous disturbance within some days or weeks. As a rule, pains and paresthesia in the feet and hands or in the legs and arms are the first symptoms, and persist during the whole course of the trouble. The *atrophic paralysis* soon follows, which also involves exclusively, or for the most part, the *distal* portions of the extremities. The *extensors* are also particularly affected in this form of neuritis, and in addition the paralysis involves more often the flexors and the small muscles of the hand than do alcoholic and lead intoxications. It generally has the character of a tetraplegia (paralysis chiropodalis). It is never entirely confined to the arms, and rarely to the legs, and is most pronounced in the latter. Electrical examination gives the reaction of degeneration with marked decrease in excitability.

The muscles and nerves are sensitive to pressure. Sensation is also almost always decreased, and the *anesthesia* or *hyperesthesia* is particularly noticeable on the feet, hands, and fingers. The pulse is generally accelerated. Increased temperature is rarely observed. *Psychic disturbances* —for instance, weakness of the memory and mental confusion—may appear in the course of the disease. Epilepsy and amaurosis are more rarely observed.

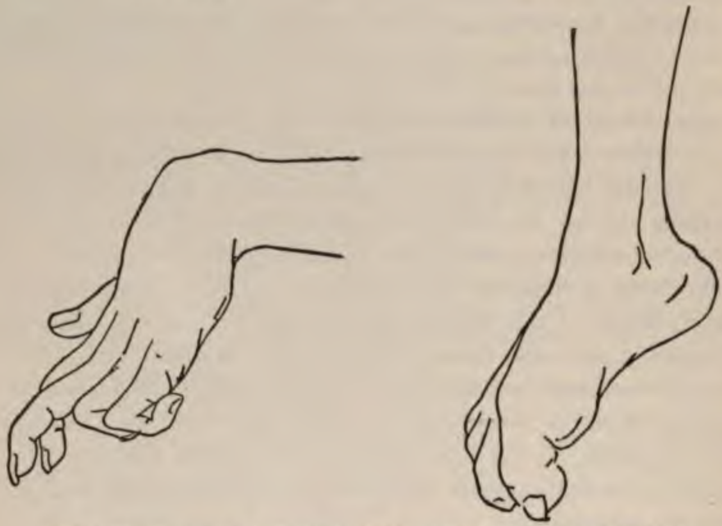
Ataxia has often been noticed. It may be the most pronounced symptom, though it is generally combined with symptoms of paralysis. A motor and an ataxic form of arsenical polyneuritis have been differentiated; as the knee reflexes are almost always absent in these cases with ataxia, they may be very similar to tabes, as Dana particularly has shown (arsenical pseudotabes).

Trophic disturbances of the skin are relatively frequent: eruptions of herpes, pemphigus, etc., glossy skin, falling out of the hair, etc. Herpes zoster has also been often observed following upon the medicinal use of arsenic. The bulbar nerves are almost always spared (Müller).

The prognosis is favorable, and the paralytic symptoms in general gradually disappear, the muscles last affected recovering their motility first, though convalescence may be protracted over months or years. In some cases secondary contractures in the antagonists of the paralyzed muscles develop. (Figs. 173 and 174.)

Life is rarely in danger. In one case pneumonia occurred and produced death; in a few cases death followed upon cardiac paralysis.

FIG. 173. FIG. 174.



Paralytic contracture in arsenical paralysis. (After Erlicki and Rybalkin.)

We know less concerning the forms of polyneuritis produced by other poisons. This is particularly true of the mercurial form. In the majority of such cases (Forestier, Leyden, Engel, etc.) syphilis had preceded the attack, and the mercury was used in the treatment of this disease; this was not so, however, in the cases reported by Kétli and Spillman-Etienne. There is no doubt that a syphilitic polyneuritis—not specific, but of syphilo-toxic origin (Schultze, Buzzard, and myself have described cases of this character; compare also the chapter on Landry's paralysis)—does occur. Notwithstanding the experimental results of Letulle and Heller, which have been questioned by Brauer, there are no observations from which we can actually determine whether mercurial polyneuritis occurs or not. It is to be noted that polyneuritis in syphilitics may be made worse by the use of mercury. Minkowski, Engle, and myself have observed this.

THE INFECTIOUS FORMS correspond for the most part to the toxic types.

Increased temperature—to 104° F.—has been observed at the beginning of the disease; it may also occur later on in its course. Delirium, splenic enlargement, and albuminuria have been occasionally noticed; also almost always symptoms indicating constitutional disturbance,—loss of appetite, headache, etc. Severe gastric symptoms—continued vomiting, diarrhea, and icterus—were present in a few cases. Profuse perspiration occurs more rarely. Concerning the symptoms of paralysis, there is nothing to add to what has already been said, though we will in the following paragraphs discuss a few types.

DIPHTHERITIC PARALYSIS.

This is the most common of the infectious forms of polyneuritis. There are a localized and a generalized form, which, however, cannot be always differentiated from each other. The milder as well as the severe cases of diphtheria may cause postparalytic conditions, which generally occur two or three weeks after the termination of the disease, but in some cases during its course. This paralysis occurs more often in children, though adults are not less in danger.

The most common form of diphtheritic paralysis does not correspond to the type of a polyneuritis. It confines itself more often to the small group of muscles which move the palatine velum. During the diphtheria, or soon after, nasal speech and difficult deglutition are noticed, fluids flowing back through the nose and solid food being swallowed with difficulty.

Objective examination shows that the velum hangs limp and does not contract during phonation. Anesthesia of the mucous membrane and loss of the pharyngeal reflex are generally present. The paralysis of the palatal muscles is generally an atrophic one. Detection of the reaction of degeneration may, however, be difficult. This paralysis disappears, as a rule, within a few weeks.

In not a few cases the paralysis has a greater involvement, extending first to the *ocular muscles,* more rarely to the muscles of the pharynx and larynx, or at the same time to all these regions.

Of the eye muscles, the ciliary is the most frequently and earliest involved. Paralysis of accommodation reveals itself through the sudden onset of ocular disturbance. The pupillary reaction is generally intact; even the reaction of accommodation may be present, despite the loss of accommodation. The abducens may be involved in the paralysis upon one or both sides; the oculomotor or the entire set of external ocular muscles may be affected. Trochlear paralysis was observed in one case (Krauss).

Paralysis of the pharyngeal muscles produces difficulty in swallowing or complete deglutitory paralysis. In these cases the muscles moving the epiglottis are at times paralyzed. Anesthesia and loss of the pharyngeal and laryngeal reflexes are observed. The epiglottis cannot be drawn down, and the danger of food passing into the trachea is great. The paralysis occasionally involves the recurrent laryngeal nerve, and produces hoarseness and aphonia. Involvement of the cardiac nerves, which, unfortunately, is not so rare, reveals itself through retardation, and later through acceleration and irregularity, of the pulse. The respiratory muscles are also often involved in the paralysis.

Even in these localized forms of diphtheritic paralysis the tendency to extend to distant nerve regions is observed, occasionally first noticed through disappearance of the knee reflexes, without any other disturbances in the extremities.

These cases form the transition to the generalized forms, which are characterized by disturbances of *motility* and *sensibility* in the *extremities*, generally following upon the above described local paralytic symptoms, which may, however, be confined to the soft palate. After the disappearance of the palatal paralysis, and after the other paralytic symptoms have partly receded, the patients complain of weakness, parasthesia, and pains in the legs or in all four extremities. These troubles increase from day to day. Disturbances of *motion*, of *sensation*, and of *co-ordination* are the objective signs of the disease.

In many cases we find particularly anomalies of sensation, especially at the distal portions of the extremities, with ataxia and uncertain gait, so that the similarity to tabes is pronounced. In the majority of cases, however, the paralysis is the most characteristic symptom. In addition to a diffuse paresis, a *degenerative paralysis*, with complete or a partial reaction of degeneration, involving particularly the peripheral parts of the limbs, is found. The degenerative character of the paralysis is, however, not always distinctly marked. Walking is pronouncedly impaired or impossible. Westphal's symptom is always, Romberg's symptom often, present. The anesthesia or the hypesthesia is likewise more pronounced upon the distal sections of the extremities; tactile sensation, the sense of position, and that of location are, as a rule, decreased; hyperalgesia may in addition be present or appear later in the disease. The skin reflexes are occasionally increased.

Disturbances of the bladder and rectum are generally not present, though they have been occasionally noticed (Katz).

These paralytic symptoms reach their height in from one to three months, the palatal, laryngeal, ocular, and pharyngeal paralyses having already disappeared. In some cases the trunkal and intercostal muscles and also the diaphragm take part in the paralysis; involvement of the glossal and facial muscles is not common. In one case, I observed facial paralysis with partial reaction of degeneration. In another case, in addition to other symptoms, the lumbar pelvic muscles were affected, so that lordosis was present, and the child in raising itself from the floor climbed up upon itself.

Hansemann in describing the history of his own case has made us acquainted with a particularly severe one. The development of the disease was preceded by a number of the most varied infections, scarlatina, repeated angina, septic infection, pleuropneumonia, etc.

Eighteen days after the onset of the diphtheria, paralysis of the soft palate and tachycardia occurred. Sensory disturbances extending over the mucous membrane of the lips, tongue, brow, and skin of the face and head then appeared. The senses of smell and taste decreased in intensity; the movements of the lips and tongue, swallowing, chewing, speech, and voice were impaired, and the ocular muscles were involved. The sensory disturbances then extended to the legs, the knee reflexes disappeared, and were followed by weakness, anesthesia, and ataxia in the lower extremities. Finally, complete motor paralysis, atrophy of many muscles of the arms and legs, and entire loss of the sense of position, girdle sensation with dyspnea, etc., occurred, and also spontaneous movements which the patient saw but did not feel. Three months after the beginning of the disorder, recovery ensued. In the examination which I undertook, four months after the onset, pronounced quantitative decrease in electrical excitability without any reaction of degeneration was present. By January of the following year he had recovered, though he still complained of paresthesia in the ulnar region.

The duration of the disease depends upon its severity and extent. The milder localized paralyses recover in a few weeks; the severe generalized forms may last for months or even a year, though this is very rare. In a case reported by Ziemssen, complete recovery only occurred eight months after the beginning of the disease, and one year afterward marked disturbances of electrical excitability, particularly a faradic reaction of degeneration, were still present. The average length of the generalized form of paralysis may be regarded as about three months.

The prognosis for the form which confines itself to the palatal and ocular muscles is favorable. Recovery can always be expected. The prognosis as to recovery in a severe case is always a favorable one, provided life can be preserved. This is in danger when the vagus is involved in the paralysis. Cardiac paralysis, unfortunately, is not a rare occurrence. Deglutitory paralysis may also cause death[1] by inanition, or, what is more common, by aspiration pneumonia. Paralysis of the respiratory muscles, particularly of the phrenic, is also a dangerous sign (Pasteur). Whenever these or other complications—as, for example, myocarditis—are not present, complete recovery may be expected.

---

[1] In an idiotic child I observed the diphtheritic palatal paralysis persist. B. Fraenkel also cites such a chronic case. In a case of generalized paralysis after retrocession of all symptoms I still observed a lost palatal reflex. A postdiphtheritic hemiplegia combined with the above described postdiphtheritic paralysis, as I observed in one case, is very rare. In this case hemiplegia occurred first, and, as the paralysis following upon it spread to the extremities, the knee reflex remained normal upon the hemiplegic side, while it was lost in the other leg.

In twenty-eight cases of this kind which I have observed, and in which I could follow the course, only four ended fatally. In these the heart was involved; in one case nephritis occurred in addition as a complication. The individuals were from three to six years old. In the adults who were affected by diphtheritic paralysis there were fourteen males and three females from fifteen to twenty-eight years old; all recovered. Where death occurs, it is to be expected early; after six weeks the danger is very slight.

Since the introduction of antitoxin treatment, our experience does not show any decrease of postdiphtheritic paralysis. Lublinski claims that the generalized form occurs more often without the localized type preceding it. That had already been observed before the serum treatment, though very rarely.

Though pharyngeal diphtheria is the rule, paralysis may occur in any other part of the body, for example, after diphtheria of the vulva, rectum, etc. (Gassicourt, Roger). I observed paralysis of accommodation occur after a diphtheria-infected wound of the forearm before a generalized paralysis came on.

In a few cases postdiphtheritic paralysis occurred without being preceded by symptoms of an infection (Boisserie, Senator, Escherich, Guthrie). Henoch doubts this, and thinks that the pharyngeal diphtheria was overlooked. In a few cases (Bourges) a diphtheritic form of paralysis was said to have developed after a simple angina.

The cases reported by Eisenlohr, of an infectious form of multiple neuritis in which diphtheria was not present, are very similar to the forms described above, in that the palatal and deglutitory muscles, which are almost always spared in the non-diphtheritic forms, were nearly always involved. Paralysis of the soft palate and of the laryngeal muscles has also been observed after erysipelas.

Beriberi (also called kakke), which occurs in the tropics, is, according to the observations of Scheube, Baelz, and others, a form of polyneuritis characterized particularly by pronounced involvement of the nerves innervating the heart and blood-vessels. Glogner claims to have found in the blood of these patients malaria plasmodia or similar structures. Polyneuritis as it occurs with us may occasionally be very similar to this type (Rosenblath).

PUERPERAL NEURITIS.

This has only been recognized in the last few years from the observations of Moebius, Kast, Lunz, Tuillaut, Mader, Eulenburg, and others. Here also a localized and a generalized form can be differentiated, the first involving preferably the median and ulnar nerves, which may, however, involve the axillary and supraclavicular nerves, or be limited to a few nerves of the lower limb. The generalized form extends over the extremities and cranial nerves and may be very similar to the type of postdiphtheritic paralysis. Optic neuritis may also occur (Schanz). It may also run a course similar to that of Landry's paralysis.

It is plain that an infectious agent (puerperal infection, septic infection) is the cause. Polyneuritis may also appear during pregnancy, particularly in those who are affected with hyperemesis gravidarum ; in some of these cases abortion is necessary.

Septic polyneuritis has not been carefully studied. Kraus has lately gathered together all that we know of it.

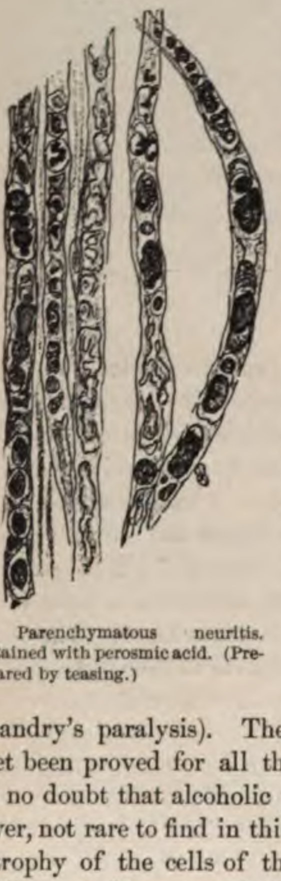
Among the forms of polyneuritis which can be referred to autointoxication, the diabetic type is most important. This is characterized by an atrophic paralysis appearing, together with severe neuralgic pains with a remittent course, generally leading to recovery. It involves particularly the crural, obturator, and peroneal regions. I have observed it in the upper extremities. The paralysis of the crural and obturator may have already disappeared before the peroneal is affected. Partial reaction of degeneration was generally found. Sensory disturbances were often present. Westphal's symptom was observed in many cases. The clinical picture (compare page 131) may be very similar to that of tabes dorsalis (diabetic pseudotabes). The prognosis is favorable, though several grave cases have occurred.

The form of polyneuritis which occurs in the aged,—senile polyneuritis,—which I described some years ago, is characterized by its insidious course and the slight intensity of sensory symptoms of excitation ; the paralysis also rarely reaches a high degree, and spares the cranial nerves. Senile polyneuritis is capable of retrogression. I have several times seen cases recover, though in general the arteriosclerosis present is a menace to life, the patient sometimes dying from an apoplectic stroke (Stein).

In a severe case of multiple neuritis caused by carcinoma, the optic nerves were affected, in addition to the extremities (Miura).

Pathological Anatomy.—Inflammatory and degenerative alterations of the peripheral nerves form the anatomic basis of polyneuritis. The peripheral branches of the nerves and the muscular and the sensory branches of the skin are the particular parts in which the neuritis develops. Signs of inflammation, as a rule, are not so marked as are signs of atrophy. In many cases swelling and redness of the nerves can be observed macroscopically, and also a hyperemia of the nerve-sheaths. Hemorrhages have been rarely noticed (Eichhorst, Dejerine).

FIG. 175.



Parenchymatous neuritis. Stained with perosmic acid. (Prepared by teasing.)

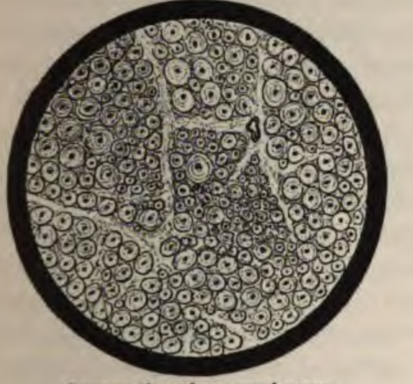
Perineuritic and interstitial alterations are either absent or slight in degree, while the nerve-fibres themselves show signs of more or less complete atrophy. All degrees from the merest trace of Gombault's periaxial neuritis to total degeneration of the nerves are found. Signs of regeneration may also be observed (Gudden). Fig. 175 shows the degeneration of the myelin, it appearing in clumps, as it is found, for instance, in the muscular branches of the peroneal nerve. Fig. 176*b* is a cross-section of a nerve in interstitial and parenchymatous neuritis (compare with Fig. 176*a*). The peroneal nerve and its branches, particularly the saphenous major, are most often affected, as well as the branches of the musculospiral nerve, etc., in involvement of the upper extremity. Close harmony generally exists between the clinical symptoms and the pathologico-anatomic observations. It is only in very acute cases that we find severe symptoms of paralysis with slight or even negative pathologic conditions (compare Landry's paralysis). The peripheral character of the process has not yet been proved for all the above-described forms of paralysis. There is no doubt that alcoholic paralysis is due to polyneuritis. It is, however, not rare to find in this form slight central alterations also,—a slight atrophy of the cells of the anterior horns, or a poliomyelitic focus, or diffuse or disseminated inflammatory conditions, as I (and also Leyden, Pal, and others) have observed in many cases. These are also produced by the action of the poison, but are too slight and incomplete to be the nucleus of the disease. The psychic disturbances, however, reveal the fact that multiple neuritis may be accompanied by involvement of the brain, as Bonhoeffer, it appears, was able to show by anatomic examination. Vagus symptoms, as well as paralysis of ocular muscles, may be referred in some cases to central nuclear processes or to hemorrhagic inflammation of the nerve nuclei (Thomsen).

Though alcohol affects particularly the peripheral nervous apparatus, evoking here the most marked alterations, nevertheless its toxic action may at the same time extend to all sections of the central nervous system.

Modern observations of polyneuritis have shown almost constant alterations in the central nervous system (Marinesco, Bickeles, Sana, Goldscheider and Moxter, Preiss, Schlesinger, and others). These were generally alterations which were produced by the same poison that caused the peripheral neuritis. Diffuse and disseminated degenerations of the roots and the posterior and anterior horns have been found. Certain alterations have been looked upon as a simple result of the peripheral neuritis, as, for instance, those obtained by Nissl's method in the cells of the anterior horn, and those observed in the posterior roots and posterior horns by Marchi's method.

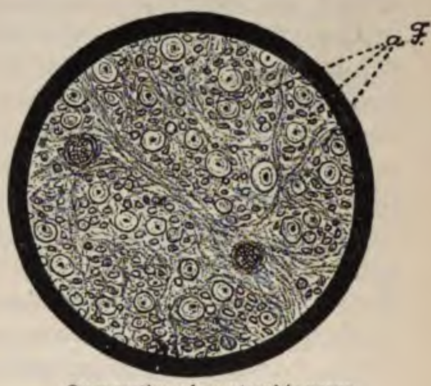
The peripheral seat of lead paralysis has been confirmed by the majority of the anatomic examinations. This poison also acts sometimes upon the spinal cord and produces severe anterior poliomyelitis, as in the case described by me. That it also extends to the brain is evident from the symptoms described of saturnine encephalopathy. The paralysis of the musculospiral (radial) nerve is of course the only constant symptom, though only part of its fibres are affected. Experiments (inspiration of lead salts) have been made on animals, and an atrophic paralysis was produced in this manner, the cause of which was a focal poliomyelitis. We know less concerning the nature of arsenical paralysis. The few anatomic examinations (Alexander, Henschen, Erlitzki, and Rybalkin) and to a greater extent the clinical nature and the course of this paralysis point to a peripheral neuritis, although in addition alterations in the spinal cord have been found, and in some cases are probably the chief factor.

FIG. 176*a*.



Cross-section of a normal nerve.

FIG. 176*b*.



Cross-section of an atrophic nerve.
a F., atrophic fibres.

The observations of P. Meyer, Gombault, Arnheim, and others make it clear that the chief basis of *diphtheritic* paralysis is a neuritic process. The diphtheritic poison acts directly upon the nerves in the vicinity of the foci of infection (palate and pharynx), from here extends to the nerve-tracts innervating the laryngeal and ocular muscles, and finally extends farther. That the toxin first acts locally upon the nervous system is clearly shown by a case in which after a diphtheritic inflammation of the navel in a new-born child, the abdominal muscles were first paralyzed. Paralysis of accommodation has also been observed as the first symptom after an intestinal diphtheria. Signs of an interstitial and parenchymatous neuritis and hemorrhages in the ocular nerves have been seen. *Nodal neuritis* has also been observed. Buhl found micro-organisms in the nerve-sheaths. Signs of arteritis have been detected in patients who died from paralytic diphtheria. We cannot assume that the diphtheritic paralysis is due to a primary myositis, though pronounced alterations in the muscles, and in one case limited to the muscles, have been described. Experimental researches which have been undertaken recently by Crocq and others have shown the neuritic process to be the result of the infection, but alterations in the roots, meninges, and particularly in the spinal cord, have been observed at the same time (myelitic foci). The anatomic examinations on human beings, carried out according to the modern methods, as also those of Sano in my laboratory, and those of Bikeles, Preisz, Katz, and others, and also older observations (Dejerine, Gombault), have revealed marked degenerations in the roots of the spinal cord and in the cord itself. In several cases, as in those of Kohts and Hasche, negative results were obtained. We cannot say with certainty whether the diphtheritic poison acts upon the whole nervous apparatus or not, the most severe disturbances appearing now here, now there, but most often in the peripheral nerves, or whether it may injure toxically certain regions without producing structural alterations in them.

The peripheral nature of diabetic, tubercular, senile, and cachectic polyneuritis is established, though complete anatomic examinations are missing for some of these forms.

It is not probable that in those cases of multiple neuritis resulting from infection, the micro-organisms themselves produce the nervous degeneration; it is generally toxic material, chemical bodies, which act similar to alcohol, lead, and other poisons when introduced into the system, and in this way injure the nervous system.

The diagnosis of multiple neuritis is in general easily established, particularly so whenever the paralysis exactly corresponds to a peripheral type. Difficulties are particularly due to two reasons: When the anesthesia and ataxia are very prominent, the clinical picture may be so similar to that of tabes dorsalis that even the experienced diagnostician is deceived. Generally, however, the acute onset, the detection of a toxic or infectious basis, the absence of bladder disturbance, the pupillary rigidity (noticed only twice in alcoholic neuritis, once by Eperon and once by myself), etc., will give sufficient data for a certain diagnosis. The diagnosis of multiple neuritis is also certain when the sensitiveness of the nerves and muscles is very pronounced, when degenerative paralysis is combined with ataxia, etc. Isolated cases of multiple neuritis have been described in which it took a chronic course, paralytic symptoms being absent, and bladder disturbances and a girdle sensation coming on, so that experienced physicians made a diagnosis of tabes. It is at least advisable in cases of pronounced alcoholism to favor a diagnosis of neuritis in doubtful cases. Mal perforant was observed by me in two cases of multiple neuritis. Gastric crises do not occur in multiple neuritis. Vomiting may at times repeat itself, but is never painful, is easily relieved, and never reaches the intensity of tabetic attacks of vomiting. Observation in a hospital, with the introduction of proper measures, particularly the withdrawal of alcoholic drinks, will help to clear up the case. It should be remembered that a spinal-cord disease can develop from alcoholism, as disease of Goll's column in a case of Vierordt.

In those cases in which the trophic motor disturbances and a degenerative paralysis are the most prominent symptoms, the disease may closely resemble *anterior poliomyelitis*, the acute as well as the subacute form. In neuritis, however, sensory disturbances are almost always present, or were present originally: severe pains, paresthesia, and at least a slight hypesthesia in the feet and finger-tips. Muscular sensitiveness to pressure may also occur in poliomyelitis, but severe pain on pressure upon the nerve-trunks is indicative of neuritis. The paralysis in acute poliomyelitis is rarely symmetrical, but is more often limited to one extremity. If the disease has developed acutely, a return of the fever in the further course, as well as a progressive appearance of new paralytic symptoms, is indicative of neuritis. The involvement of cranial nerves is so rare in poliomyelitis that this observation in dubious cases should be referred to neuritis. Neuritis is positively present if inflammatory alterations are found in the optic nerve.

Raymond does not believe that polyneuritis, poliomyelitis, and Landry's disease can be sharply differentiated, as they have the same etiology. He acknowledges, however, that a differentiation is advisable for clinical reasons.

Eisenlohr has called attention to the fact that *trichinæ* may cause symptoms similar to those of neuritis (and myositis), evoking pains, paralysis, and edema; trophic disturbances of the muscles with reaction of degeneration and even Westphal's symptom can occur from this cause. The diagnosis may generally be established from the development: gastric disorders have preceded; fever and severe muscular pain follow; then the characteristic muscular swelling comes on, the difficulty in moving the eyes, with edema in the vicinity, the disorders of speech, voice, dyspnea, etc. The relationship between neuritis and polymyositis is given in the discussion of the latter.

Treatment.—The treatment of multiple neuritis is one of the most successful in neurotherapy. The cause of the trouble must first be established and the injurious factors removed. The withdrawal of alcoholic stimulants is necessary in every case, except where cardiac weakness is present.

In lead poisoning all material containing lead must be avoided; the body, particularly the teeth and nails, must be kept clean; the clothes worn at work must be removed afterwards, etc. The removal of the lead from the body is secured by the use of iodide of potassium, frequent alvine discharges (laxatives, sulphur salts), diuretics, and baths, especially sulphur baths.

If the source of the poison is endogenous, it may be removed at times by the cleaning out of putrid, septic masses, the action of *diaphoretics*, etc. Diabetic neuritis does not always disappear with the cessation of glycosuria, but the fact that the neuralgias lessen in severity when the diet does not comprise saccharine food demands a strict *antidiabetic diet* to combat this form of neuritis. Malarial neuritis is generally cured by quinine.

If the polyneuritis is due to syphilis, a careful mercurial treatment is appropriate. It may, however, fail to do good, or may even do harm. In such cases use immediately the other measures recommended for the treatment of polyneuritis.

In the treatment of beriberi, change of climate does good.

In all forms of neuritis a *strengthening diet is important*. Milk, and foods made from it, meats, and fat (perhaps cod-liver oil) should be given at frequently repeated meals.

In diphtheritic paralysis of deglutition one must seek to prevent the entrance of particles of food into the air-passages, and to keep up the strength of the patient by *feeding through a sound*, and, if necessary, administering nourishing clysters. Ziemssen recommended the introduction of the following, four times a day, through a sound: One-half to one litre milk soup with fine flour, two to four eggs, sugar, and port wine, stirred together, varied with one-half to one litre of concentrated meat-broth, to which four teaspoonfuls of meat-juice, two to four eggs, and one glass of port wine have been added. Other nutritive preparations, as somatose, may be added to the fluid. If vomiting follows the entrance of the sound, the patient should be laid upon the stomach, with his head hanging out of the bed. Cardiac weakness demands the use of *stimulants*,—wine, cognac, subcutaneous injections of camphor (camphor 1.0 to 5.0 of ol. amygd. dulc., a syringeful several times a day). Faradization of the cardiac region is also recommended. If bronchitis without sufficient expectoration be present, artificial support of the expiration by pressure upon the lower thoracic parts may produce brilliant results. Bloodletting is never permissible.

Diaphoretic measures are especially advisable in the first stage of polyneuritis. Whenever the patient's strength permits it, the sweat-glands may be stimulated by the cautious use of hot baths; in other cases—particularly in existing cardiac weakness—it can be done by packing in damp sheets and woollen blankets, with the simultaneous use of hot drinks. Diaphoresis can be continued for one to two hours. I have had *good results* by this plan of treatment in severe cases also. Caution must be exercised in weak individuals; in some cases the sweat cure can only be borne if undertaken every second day.

Internal treatment can in general be disregarded, though the *salicylate preparations* and *salol* can be administered with propriety in the infectious forms of polyneuritis. If the pains are severe and cannot be relieved by the applications of warm cloths or *Priessnitz's bandages, antipyrin* should be tried, and if this fails, *morphine* should be used. I have, however, always been able to forego the use of morphine.

In the first stage *absolute rest* in bed in a comfortable position is demanded; over-exertion of the diseased nerves, even if only done once, can produce a material change for the worse. Passive movements should also be avoided, if possible. This precautionary measure is especially advisable in diphtheritic paralysis whenever the signs of cardiac or vagus disease are present. The patient should *never sit up in bed*. Even involvement of the pharyngeal and laryngeal branches of the vagus requires the exercise of great caution, as cardiac paralysis may come on quickly and unexpectedly. The room must be well aired; the patient's bed might be carried into the open air.

In alcoholic paralysis, treatment in a *hospital* is preferable to that in a private house, as only then can a patient be satisfactorily watched and alcoholic abstinence be carried out. I regard *isolation*, however, as advisable, particularly in large hospitals, in order to protect these sensitive patients from catching some infectious disease. The patient should be guarded from refrigeratory influences.

Whenever the neuritis has reached its acme, or the condition has become stationary, or the initial signs of improvement are observable, an *electric* and *mechanic* treatment is in place. As long as symptoms of irritation are still present, *stabile galvanic* treatment is advisable; later, *labile* and *faradic muscle stimulation*. In many cases, in which the electrical current was used without any result, I observed an almost instantaneous improvement from *massage*. This must be used carefully according to individual sensitiveness. Commence with mild rubbing and stroking in the neighborhood of the affected nerves and muscles, and increase the manipulations in intensity very gradually. Active and passive gymnastics can do much good, though over-exertion should be avoided as far as possible. If the paralytic condition is stubborn, subcutaneous injection of strychnine (.001–.003, or $\frac{1}{60}$ to $\frac{1}{20}$ of a grain, several times daily) is often beneficial. Injections of carbolic acid (two per cent.) or carbolmorphine in the neighborhood of the affected nerves have also been recommended.

In the later stages *baths* are of benefit; not those of high temperatures, but mild ones of 26°–28° R. (90°–95° F.). If the social relations permit, and the transportation of the patient is not accompanied with danger, a stay at Nauheim, Oeynhausen, Kreuznach, Wildbad, Wiesbaden, Teplitz, etc., is advisable, but only when improvement is already far advanced. They are particularly good for after-cures. Mud baths can also be used. In winter, a sojourn in the South may prove beneficial. During *convalescence, hydrotherapeutic measures* do good; they should be limited, however, to local applications of damp cloths to the extremities, and should be commenced at a mild temperature.

The development of paralytic contractures should be counteracted early, particularly in youthful individuals; as soon as foot-drop is indicated the pressure of the bedclothes must be removed from the feet by a wire basket, and a heavy sack of sand be so placed that it forces the tips of the toes upwards. If contractures are already present when the patient comes under treatment, they should be treated according to the usual well-known methods; tenotomy is rarely necessary.

LANDRY'S PARALYSIS. ACUTE ASCENDING PARALYSIS.

In 1859, Landry described the following characteristic symptom-complex. There develops in individuals, healthy until that time, preceded by slight subjective prodromes (general malaise, paresthesiæ in the extremities, etc.), a flaccid paralysis of the legs, generally first in only one, but extending to the other within some hours or a day. After the paralysis of the legs has become complete, it extends to the truncal muscles, and within some days reaches the arms, where the paralysis is likewise of a flaccid nature. Afterwards, the deglutitory, articulatory, and respiratory muscles are involved, and death comes on in a few days or weeks under symptoms of suffocation, except in milder cases, in which the symptoms recede in such order that the muscles affected last recover first. According to Landry's description, slight sensory disturbances belong also to the clinical picture, but the *absence of muscular atrophy* and the electrical symptoms of the same, together with the *negative result at autopsy*, were especially emphasized.

The course can be modified in that the bulbar symptoms appear first, then the paralysis of the arms, etc.

Although later observers (Pellegrino and Levy, Westphal, Kahler and Pick, Bernhardt, Ormerod, Eisenlohr, and others) have in general confirmed Landry's observations, and although not a few cases have been described which fit into his description, further observations have shown that he limited it too closely, that many cases deviate widely from Landry's type, and that the only criterion for all cases is that a flaccid paralysis develops affecting *in rapid succession* the muscles from below upwards,—*i.e.*, from the legs over the trunk and arms to the bulbar nerves (or *vice versa*, though more rarely, from above downward).

Symptoms.—The following has been established concerning the symptomatology:

The paralysis in most cases begins in one lower limb or in both. Parasthesiæ may exist at the same time. The paralysis develops rapidly, and may increase to complete paraplegia within one or more days. It is a *flaccid* paralysis, generally combined with *loss of the knee-reflexes* and skin-reflexes. Pains are entirely absent, or only appear on pressure upon the muscles and nerves, as well as upon passive movement. It is only rarely that complaint is made of spontaneous pains. The paralysis in a few days extends upward, first to the pelvic muscles, and then to the abdominal, spinal, shoulder, and thoracic muscles. It soon reaches the arms, which are likewise completely paralyzed. Finally, the labial, glossal, pharyngeal, palatal, and respiratory muscles are involved. Speech becomes indistinct and heavy; swallowing becomes difficult; pronounced respiratory disturbance comes on, simple acceleration, or Cheyne-Stokes's breathing, the signs of diaphragmatic paralysis ensue, and under *asphyxic* symptoms death ends the scene on the eighth to the tenth day, more rarely on the third or fourth, and only exceptionally after some weeks. In some cases, however, the symptoms recede in the manner described above; convalescence then may extend over a long time. If the disease commences with bulbar symptoms, death may result before the paralysis has extended to the extremities. Only rarely are the arms involved first or all four extremities at the same time.

The *sensory* conditions vary greatly. It can be regarded as the rule that gross disturbances, particularly a complete loss of sensation, are absent; but a slight decrease of sensation is frequent, either for some or all qualities of sensation on the ends of the extremities. Retardation of sensory impressions was found several times.

The muscles generally retain their *normal volume*, and react promptly to the electrical current. This is particularly true of the rapid lethal cases, and has been noticed in some of longer duration also. But in other cases, which in every other respect correspond to this type, *muscular degeneration* with varied disturbances of electrical excitability has been noticed: a quantitative decrease or a partial or complete reaction of degeneration. In one case I observed a peculiar condition of electrical excitability: the nerves and muscles reacted to weak electrical stimulation, but increase of the current strength did not increase the intensity of the contractions, their minimum and maximum lay very close together, and even the strongest current failed to secure a pronounced contraction; indeed, several times a strong current produced no contraction at all, or a weaker one than that evoked by the weaker current. Examination of a piece of muscle which was cut out revealed a waxy-like degeneration. These phenomena vanished with recovery.

The *bladder* and *rectal functions* are, as a rule, not disturbed, though there are exceptions, even complete sphincter paralysis having been observed.

The cranial nerves, not hitherto mentioned, are not involved; only in a few cases was vascular paralysis (diplopia, accommodation paresis) observed. A unilateral or bilateral *facial paralysis* has been noticed several times.

The *sensorium* is generally not involved; only when high fever and signs of a general septic infection are present is consciousness clouded.

The temperature is in the majority of cases *normal*, though periods of pyrexia have been observed in some cases. Profuse perspiration is often described.

It is desirable to discuss **the etiology** and **pathological anatomy** together. Landry thought that this disease was due to poisoning. This view has been adopted by almost all authors, and was established on a scientific basis by Westphal. The following factors point to a toxic or infectious origin. In many cases *splenic enlargement*, inflammation of the lymph glands, hemorrhagic foci in the lungs and bowels, and *albuminuria* have been noticed. The observations of Baumgarten, Curschmann, Centanni, Eisenlohr, Remmlinger, Oettinger and Marinesco, Marie and Marinesco, and Bailey and Ewing have been of particular importance. In the case described by Baumgarten, *anthrax* was probably present, and the corresponding bacilli were found in the blood and in the tissues. Curschmann described a case in which (and typhoid had not preceded it) the characteristic intestinal alterations of typhoid were found, and *typhoid bacilli* which could be grown in pure cultures were discovered in the spinal cord. Centanni found, in addition to interstitial neuritis, bacilli in the endoneural lymph spaces. Eisenlohr has lately found different kinds of bacilli in a case of Landry's paralysis, and in another, not strictly coming under this head, he has been able to determine different kinds of *staphylococci* in the central nervous system, and to refer the symptoms to *a mixed infection*. Remmlinger found the *streptococcus longus*, Marinesco *diplococci*, partly enclosed in leucocytes; in the case examined with Marie he found a micro-organism similar or corresponding to the anthrax bacillus, which was particularly abundant in the blood-vessels. In a case of acute bulbar descending paralysis, which no doubt belongs in this category, J. Seitz found the Fraenkel-Weichselbaum bacterium present in large numbers in the brain-stem and the spinal cord following the lymph tracts of the tissues. The nervous elements were unaltered. The author thought that the micro-organisms had found their way to the brain through the posterior nares.

Very indefinite and varied are the pathologico-anatomic lesions described in cases of this disease. In many cases the anatomic examination was negative, corresponding to the claims entertained by Landry, Westphal, and others. Later observations were those of Ormerod and Prince. In some cases *disseminated foci of inflammation* were found in the medulla oblongata (particularly in the pyramidal tracts), or exudates and capillary hemorrhages; in other cases similar *alterations* in the *spinal cord* (Eisenlohr, Schultze, Kétli, Hlava, Immermann, and others). Several times was a swelling of the axis-cylinders in the white substance of the anterolateral tracts particularly noticeable; in other cases there was present the slightest degree of poliomyelitis. In one case a degeneration of the anterior roots was the only alteration, in another a small *myelitic focus* was found in addition to peripheral neuritis.

Since attention has been directed to the peripheral nervous system, *neuritic* processes have been detected many times, and have been regarded as the foundation of the disease by some authors (Dejerine and Goetz, Nauwerck, Barth, Ross, Putnam, Klumpke, and others).

A combination of neuritic and myelitic processes has also been found (Krewer and others).

On the whole, late researches, founded upon the new methods, have almost all given positive results; and particularly alterations in the spinal cord, especially of the gray matter. They were either inflammatory and vascular conditions (arterial disease, hemorrhage, exudation, thrombosis, softening, infiltration, etc.), or alterations of the nerve-cells, which rarely, however, reached the degree of distinct atrophy. Such anomalies have been particularly described by Marinesco, Bailey, and Ewing. But the finer the method is, the more caution must be exercised in the valuation of observations.

It is plain that a nosologic conception of this disease cannot be placed upon a pathologico-anatomic basis, inasmuch as researches and observations of this character have varied so greatly. The clinical picture is sufficiently clear and characteristic, and there is no doubt that it is due to infectious causes and *toxins*, which in most cases are *bacterial products*. The disease may develop after diphtheria, typhoid, variola, anthrax, influenza, pneumonia, whooping cough, puerperium, gonorrhea (?), probably also septicemia and other unknown infectious processes. It seems to be able to occur in rabies or following upon Pasteur's treatment (Rendu and others). One of my patients, a hostler, became stricken with Landry's paralysis after receiving a wound from the hoof of a horse suffering from septicemia. In another case, only severe diarrhea had preceded. We certainly cannot speak of a specific infectious agent in Landry's paralysis.

Alcoholism and, particularly, syphilis have been considered etiologic factors.

Whether the entrance of the micro-organisms into the spinal cord, medulla oblongata, and peripheral nervous system is able to produce the disease, is doubtful. It is probable that the poison injures the motor conducting tracts of the spinal cord, medulla oblongata, and peripheral nerves, so that it evokes paralysis without exciting any recognizable lesions in the nervous apparatus, but that it occasionally leaves visible (microscopically detectable) lesions, found in the motor tracts of the medulla, or in the peripheral nerves, or in both places. As a rule, it touches the trophic centres and tracts so slightly that muscular atrophy does not result. We can not, however, lay down any set rules in this respect.

Gowers believes that the net of fibres in the gray matter of the anterior horns, the terminal extensions of the pyramidal tracts, are particularly involved. We would then have an interruption of the motor conducting tracts without trophic disturbances and spastic phenomena: but this is an unsupported hypothesis.

In the cases corresponding exactly to Landry's type, we have a type of disease that is easily separated from other well-known clinical pictures. The cases deviating from it closely resemble *multiple neuritis*, a disease which, in common with Landry's disease, is of toxico-infectious origin. It is not proper, however, to regard them as perfectly identical, and to make of Landry's disease a polyneuritis acutissima. There is less justification in classing it with acute poliomyelitis, though we concede with Raymond their close genetic relationship. The differentiation into three distinct forms, a bulbar, a spinal, and a peripheral, is clinically impossible.

Lately affections similar to Landry's paralysis have been produced in animals by the introduction of micro-organisms into the blood (Thoinot and Maselli).

The prognosis in regard to life is very unfavorable. Death is especially to be expected in the violent cases with rapid extension to the medulla oblongata. But a fatal ending can occur in cases of less rapid course, even after weeks. Chances for recovery are particularly present when a part of the symptoms, especially the bulbar symptoms, have receded. In two of the four cases which I had an opportunity to treat, complete recovery was secured, the course having been somewhat protracted; in the two fatal cases, it was very stormy (in one, pneumonia was the cause, in the other, tuberculosis).

Treatment.—Various remedies have been recommended. A revulsive treatment has been especially praised, particularly the use of a *cautery* upon the spinal column. It seems rational to institute a diaphoretic and antiphlogistic treatment in certain cases.

In some, in which a syphilitic infection has preceded, *mercurial* treatment has been of curative influence. *Ergotin* has been regarded as efficacious (ergotin 1.25, cinnamon water 6.0, a teaspoonful hourly); in very severe cases recovery has been claimed through this medication.

The future will show whether blood-serum therapy is practicable in this disease.

ACUTE AND CHRONIC POLYMYOSITIS.

Our knowledge of this disease is of recent origin; observations have been as yet very few. The first complete ones are those of Wagner, Hepp, and Unverricht. Other forms of myositis, particularly interstitial myositis resulting in induration, were known to older authors (Froriep and others).

The disease can occur at every age. Males are affected, it appears, more frequently than females.

It is probably an *infectious* disease, though the infectious agent is not yet known. *Tuberculosis* is a relatively frequent complication. Myositis has been observed several times in the *puerperium*. It can also develop after influenza, angina, and acute articular rheumatism. Purulent processes, as A. Frenkel showed, such as tonsillar abscesses and otitis media, can be the starting-point of the disease. This author is opposed to a differentiation of purulent and non-purulent forms, as the latter, as a rule, is also a metastatic or septicopyemic muscle affection.

The disease generally come on *acutely*, though not suddenly. General symptoms of ill health open the scene. The patients feel listless, experience a dull pain in all limbs; headache, vertigo, and gastric symptoms come on. From the beginning, or in a few days, *pains* occur whose seat is in the *muscles* of the extremities and trunk. They are described as drawing and irritating, cause a limitation of active movements, the affected muscles become gradually *incapable of functionating*, and the patient lies paralyzed and helpless in bed, without being able to move a limb.

The proximal part of the extremities—particularly the muscles of the *shoulder and upper arm*—are generally more markedly affected than the distal portions, so that the fingers can still be moved though the shoulder- and elbow-joints are lost to all movement. The muscles are *very sensitive to pressure*, even passive movements evoking pain.

Swelling of the muscles, as well as of the softer tissues and the skin over them, is one of the most important objective signs; the latter—the edematous infiltration of the skin and the subcutaneous tissue—may be so pronounced as to prevent any decision as to the condition of the muscles. These swellings are found over the muscles which are most affected, upon the truncal sections of the limbs, in the region of the shoulder, upper arm, and elbow-joint, thigh, etc., this uncommon localization of the swelling being particularly characteristic. The arms are, as a rule, more markedly implicated than the legs. Wherever the muscles can be palpated, they feel at times hard, at other times soft and flabby, even a kind of pseudofluctuation and circumscribed edema has been noticed. The skin is generally *reddened*, at times hot to the touch, the redness occasionally simulating erysipelas, being particularly marked on the face. Roseolar and urticaria-like exanthems have been noticed: Unverricht therefore speaks of a *dermatomyositis*. *Hyperidrosis* is often present.

The muscular affection rarely implicates the *respiratory* and *deglutitory muscles*,—those of the tongue, maxillary region, diaphragm, heart, and medulla oblongata. The extension of the myositis to the *respiratory muscles* causes a more or less pronounced impairment of breathing; the ingestion of food is made difficult or impossible in case the deglutitory muscles are involved. Ptosis and paralysis of the ocular muscles have also been observed. On the other hand, in mild cases the myositis may limit itself to the muscles of one extremity, to one or both thighs, to one arm, or even to one muscle. *Stomatitis* and *angina* were present in many cases.

The *sensorium* remains free, the nerves of special sense functionate normally. *Sensibility* does not appear to be affected in typical cases.

An electrical examination, on account of the pronounced edema, is very difficult to make; the result is *quantitative decrease* of excitability (particularly the direct), finally amounting to complete loss, without any qualitative changes.

The knee-reflexes are decreased or entirely absent, if the corresponding muscles are involved; the *skin reflexes* may be normal.

The *temperature* is elevated during the whole course of the disease; it may be over 104° F. The pulse is correspondingly accelerated, but tachycardia, independently of the fever, has been noticed many times. Hemorrhages from internal organs, particularly intestinal hemorrhages (Buss), have been noticed. In chronic cases the temperature elevation is not constant.

The disease may last for weeks, months, or even from one to two years. There is then an acute and a chronic course. It may terminate in recovery within some weeks, or end in death after weeks or months.

Asphyxia and aspiration pneumonia are the most common causes of death. A nephritis can also complicate matters and cause death. In a case observed by me the pains localized themselves first in the joints, without causing any edema there, then sprang to the muscles and established themselves there. The edema developed later. In two other cases, which I had an opportunity to treat during the influenza epidemic, the disease confined itself to one extremity.

If the disease takes a protracted course, *remissions* may occur.

The prognosis in regard to life is very grave, though recovery is not excluded, and is most likely to occur in cases which were mild from the beginning. There is no doubt that the observations of cases with a mild, favorable course will increase whenever we are more conversant with the clinical picture. This expectation (see first edition) has been realized, as numerous cases with a favorable outcome have been reported in the last few years (B. Levy, M. Levy-Dorn, Herz, and others).

The anatomic basis[1] is a *parenchymatous* and particularly an *interstitial myositis*, implicating the whole musculature of the body or a large part of it. The disease can generally be recognized macroscopically by the edema, discoloration, and imbibition of the muscles, which are not rarely also the seat of hemorrhages. Fig. 177 illustrates a microscopical cross-section of a case of this kind, which shows very pronouncedly the round-cell proliferations in the interstitial tissue.

Differential Diagnosis.—It may be confused with *trichinosis*. The clinical pictures are so similar that polymyositis has been described as pseudotrichinosis. Trichinosis affects, however, a number of persons who have eaten meat of the same animal. Gastric disturbances, also, are prominent symptoms, and at the beginning are dominant. The ocular, masticatory, and laryngeal muscles are chiefly affected, and are the seat of severe pains, while edematous swelling of the face and eyelids develops at an early date.

---

[1] We will not enter into a discussion here of traumatic purulent and interstitial myositis, a disease particularly familiar to surgeons. B. Laquer described an acute interstitial monomyositis of a remittent type with an unknown etiology.

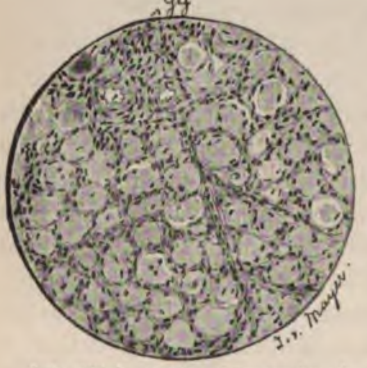
Mild cases can be confused with muscular rheumatism, though the swelling of the muscles, the discoloration of the skin which is frequently present, etc., should readily serve to distinguish them.

The symptomatology of polymyositis has likewise much similarity with that of *polyneuritis*; a combination of both diseases, a *neuromyositis*, as Senator particularly has shown, is frequently seen. They differ from each other in that in neuritis sensitiveness of the nerves to pressure and sensory disturbances are almost always present, and the paralysis is accompanied by a reaction of degeneration, while the muscular swelling and edema are not prominent symptoms.

Syphilitic myositis can also correspond to the picture of polymyositis and be very similar to it (Herrick). It is noteworthy that myositis ossificans may also run a progressive course with fever, in that the ossifying process follows each attack.

FIG. 177.

94



Interstitial myositis. Cross-section of muscle.

Some observations (Schultze, Oppenheim-Cassirer) indicate that there are forms of this disease in which the affection terminates in progressive or stabile muscular atrophy.

There is also a form of myositis which is confined to a *few muscles*, and which is caused by over-exertion. Strümpell observed it, for example, in the lower limbs of an organ-player who had used the pedal for hours. I saw it appear in a man after some severe marches. The prognosis of this form, which should be separated from the infectious, is favorable.

Treatment is in general similar to that of neuritis. In a case observed by me, salicylate preparations and wet packs had a favorable influence. For the later stages, massage and gymnastics are especially in place.

CRAFT PALSIES; PROFESSIONAL PARESES.

In certain occupations which necessitate long-continued over-exertion of certain muscles, there sometimes results an *atrophic paralysis* of these muscles. The small *muscles of the hand* are, for evident reasons, most frequently implicated. Closer analysis of the cases shows that it is not only over-exertion that is in play, but frequently, also, the *pressure* exerted by the workman's tools upon the nerves and muscles. Cases of this kind have been described by Berger, Remak, myself, and others.

In this way there occurs in *platers* an atrophy of the *first interosseous* and the *opponens pollicis;* the same is noticed in *filers*, *paper-glazers* (in whom a toxic factor, lead, is also present). In *locksmiths*, *cabinet-makers*, *blacksmiths*, *gold-polishers*, and other manual laborers who labor day after day with a plane, bore, or other instrument in a similar manner, atrophic paralysis of the small muscles of the hand is not a rare occurrence. Ulnar paralysis has been observed from over-exertion in *rowing*, also in *glass-workers*, who had supported the inner surface of the elbow-joint upon a high table, as also in xylographers (Bruns). Atrophy of the ball of the thumb is said to have resulted from exertion in writing and zither-playing. Atrophy of the *interossei* as a result of *cigar-rolling* (Coester), paralysis and atrophy of the muscles innervated by the median as a neuritic complication of milker's cramp, etc., also occur. In drummers a paralysis of the extensor pollicis longus, in which also the small muscles which extend the terminal phalanx of the thumb generally take part, more rarely a paresis of the flexor pollicis longus (Bruns, Zander), has been noticed. In a man who worked at a bookbinding press, and who had to grasp the handle constantly with his hand, I observed an atrophy of almost all the hand or finger muscles. A weaver, whom I treated, had the symptoms of paralysis of the right triceps muscle, due to the fact that he had to extend his forearm about twenty thousand times a day.

Craft palsies occur more rarely in the lower extremities, though a part of the paralytic conditions in the peroneal and posterior tibial regions, occurring in workmen who labor in a kneeling position, as in *potato-pickers* (Zenker), *seed-sowers* (Hoffmann and others), etc., belong here. In addition to over-exertion, the pressure exerted upon the nerves when the knee is flexed acts injuriously. Laceration of the nerves may also play a part.

The professional pareses are not rarely accompanied by slight *pains*, *paresthesias*, and *sensory disturbanes;* this is explainable only by assuming that the pressure which affects the peripheral nerves evokes a *neuritis*. Whether the muscular atrophy is merely a result of this neuritis or is a direct effect of over-exertion, cannot always be exactly determined.

Alcoholism, lead intoxication, marasmus, etc., favor the onset of these pareses. There are cases of this kind in which we must speak of a toxico-professional paresis. In a young lady treated by me, atrophy of the small muscles of the hand came on just as she began to recover from an attack of *influenza*. An acute anterior poliomyelitis of childhood leaves behind, according to our experience, a disposition to these forms of atrophic paralysis. I treated a man, for instance, who found it necessary to support himself on and firmly grasp a cane with his right hand on account of poliomyelitic paralysis of the right leg; there resulted from this an atrophic paralysis of the muscles of the right hand, which receded completely under careful rest and electrical treatment in the hospital.

These conditions generally develop in a chronic or subacute manner; several times, however, I was able to determine an acute or step-like onset.

The prognosis of these artisan's palsies is in general *favorable*. If the patient takes care of himself in proper time, so that all injurious factors are removed, the muscles implicated almost always recover. Only in rare cases does the disease seem to be the starting-point of a progressive muscular atrophy.

Treatment consists of perfect rest and sparing of the muscles involved. The galvanic current can be used in addition.

THE NEURALGIAS.

General Considerations.—We understand by neuralgia a severe paroxysmal pain, experienced in the tract of a nerve-trunk or its branches. The patients generally point out the direction of the pain with the finger, as it is not the rule for it to limit itself to a point or spread out over a plane surface. Only in rare cases in which it has its seat in the terminal branches of the nerves is, instead of a linear extension, a point-like or surface extension noticed.

Neuralgia can come on at any age, though it is rare in childhood and occurs mostly in persons of middle age. According to Bernhardt males are affected more often than females. *Puberty, pregnancy*, the *puerperium*, and the *climacterium* increase the disposition.

The causes are numerous. One of the most important is a *neuropathic disposition* (Anstie). Hereditarily weak and nervous individuals are the most easily affected.

Exhausting diseases or loss of blood and secretions can also lay the foundation for this evil; *anemia, cachexia, senility*, and *arteriosclerosis* particularly form a favorable soil for it. Obstipation may also favor its onset.

Infectious diseases may help in evoking neuralgia through the exhaustion produced by them, and also by bringing into the blood *toxic material* which injures the nervous system directly. Malaria-neuralgia can, without doubt, be referred to a virus. It is probable that the neuralgia occurring after typhoid, smallpox, influenza, and other infectious diseases is due to a similar cause.

Whether a true neuralgia occurs from syphilis, as Fournier, Obalinski, and others assume, is doubtful. A syphilitic or gummatous neuritis may clothe itself in the form of a neuralgia for a long time.

The etiologic importance of many *poisons* has been established. Lead, mercury, copper, alcohol, nicotin, arsenic, and others are among the causative factors of neuralgia. The forms developing in diabetes mellitus, and more rarely in arthritis, can also be due to chemical bodies. This is indicated by the fact that the intensity of the pain is occasionally proportional to the quantity of sugar excreted, and that arthritic neuralgia may suddenly disappear after an attack of gout.

Refrigeration, mental and physical *over-exertion*, and *emotional disturbances* are also given as causes of neuralgia, generally as mere exciting causes. Over-exertion of the ocular muscles and refraction anomalies are given an important place in the etiology of neuralgia by some authors (Stevens). The *rheumatic* diathesis favors the onset of neuralgia; neuralgia and rheumatism may also occur conjointly.

Injuries to the nerves and their branches, particularly shot-wounds (Mitchell, Keen), laceration and compression by scars, tumors, aneurisms, varices (Henle, Quenu), often evoke neuralgic pains, though it is just these cases in which it is difficult to separate true neuralgia from neurotic-degenerative diseases of the nerves. Those cases in which an irritation in the periphery—be it from a cicatrix, from a bad tooth, from a connective-tissue inflammation, or from some disease of an internal organ—produces neuralgic symptoms are justifiably styled true (idiopathic) cases.

Pains of a neuralgic character occurring in the course of organic nervous diseases, tabes, syphilitic meningitis, tumors at the base of the brain and of the spinal cord, etc., must be differentiated from neuralgia.

Symptomatology.—The pains come on paroxysmally, the individual attacks lasting from one or several minutes to several hours. But even in the attacks the pain is not continuous, but comes on in jumps, which are described as cutting, boring, burning, etc. They do not generally disappear as rapidly as lancinating pains, but last several seconds or minutes. In the intervals pain is entirely absent, only occasionally a dull feeling of pain is present, and the paroxysms consist in exacerbations of it. They occur spontaneously or are excited by *movements* of the parts of the body in which the neuralgia has its seat, by touching the same, by the influence of cold air, by the ingestion of food, by emotional excitement, or by coughing, straining, sneezing, etc.

The attack is frequently accompanied by *secretory, motor*, and *vasomotor symptoms*. Watering of the eyes, salivation, local hyperidrosis, twitchings (for example, convulsive tic in trigeminal neuralgia), paling or, more often, reddening of the skin, are generally accessory symptoms. Circumscribed edema is more rare. In cases of longer duration, swelling and infiltration of the skin and soft parts, even of the periosteum and bones, occur. A stabile edema, a chronic dilatation of the blood-vessels of the integument, erythema, etc., are occasionally noticed. The relationship between herpes and neuralgia needs no special description. Falling out and graying and other alterations in the color of the hair are rare phenomena.

Paralysis does not occur in neuralgia, though the pains often cause a limitation of active movements, and thereby a kind of pseudoparesis. A simple muscular emaciation, due, probably, to deficient blood-supply, occurs (Nothnagel).

Some of the symptoms, particularly the motor symptoms of irritation, are no doubt of reflex origin.

The pain has its seat in the main trunk of a nerve or in several or all of its branches. At the height of the attack it may extend to the branches which are not involved at other times or to other nerves. More rarely the same nerve on the opposite side becomes implicated at the acme of a paroxysm. A kind of *transfer*, a springing of the pain to the other side —spontaneous or as a result of electrical or operative treatment—occurs occasionally as a transient phenomenon. The skin in the affected area is often hyperesthetic, particularly to slight touch, while deep pressure may ameliorate the pain. More rarely, and generally only in later stages, there develops a slight hyperesthesia (never anesthesia) in the area innervated by the diseased nerves (Nothnagel).

In many cases *pressure-points* in the course of the affected nerves are found, on which pressure causes pain. These, first described by Valleix, correspond generally to the places of exit of a nerve-trunk from a bony canal or a place where it can be pressed against a hard floor (bones, ligaments). It is uncommon to find the nerve sensitive to pressure along its whole course. Pressure-points are often found along the vertebral column, corresponding to the origin of the diseased nerves (Trousseau).

At the height of an attack, vomiting and *retardation* of the *pulse* may occur, though this is very rare.

When the disease is of long duration, the *general constitutional con-*

dition suffers. Digestive disorders and loss of appetite occur, nutrition is impaired, even a pronounced *cachexia* can develop. The mind is likewise affected: simple depression, irritation, loss of desire for work, are common results of neuralgia; only rarely do they increase to marked psychoses, particularly melancholia. In severe, stubborn cases the disease not rarely causes thoughts of suicide.

Some forms of neuralgia are characterized by particular characteristics. Those due to malaria have a *typical*, periodical course. The attack occurs at certain hours, ceases again at a certain time, to return at an interval of from one to fourteen days. While malaria-neuralgia does not always show this intermittence, simple neuralgia may, on the other hand, sometimes take such a typical course. I have noticed this especially in cases of hysterical neuralgia. There is also a *nocturnal neuralgia.*

Malaria-neuralgia involves particularly the *supra-orbital*, *sciatic*, and *occipital* nerves. The neuralgias of diabetic origin affect mostly the trigeminal, particularly the third branch, and the *sciatic;* in addition, their symmetrical extension is also characteristic. In arthritic cases the trigeminal and sciatic nerves are also most often implicated.

A neuropathic constitution seems to increase the predisposition to neuralgia, particularly in the supra-orbital and intercostal nerves.

Influenza-neuralgia seems to seek out the supra-orbital nerve.

Nature of Neuralgia.—We do not know anything certain about the *anatomic basis* for this disease, and it would be justifiable to place this chapter under the rubric of the functional neuroses. The fact that a neuralgia may be present for years, for even a decade, without causing any material lesion of the nerves, seems to be opposed to the idea of considering it an organic disease. On the other hand, a mild *neuritis* may run its course under the picture of a *neuralgia*, and in some forms of neuralgia, particularly sciatica, certain symptoms come on, often early in the disease, which we are wont to refer to neuritis.

At any rate, on account of typical types and forms, we must separate neuritis from neuralgia, conceding that the border is not a sharp one, and that there are cases in which it cannot be determined with certainty whether neuralgia or neuritis is present. It is probable that minute *disorders of nutrition in the nerves*, in their sheaths, or in the nervi nervorum form the basis, and that under certain conditions they increase to *neuritis* and *perineuritis*. That the pain in many cases begins in the roots and the central origins of the nerves is also possible.

The view of Oppenheimer, that the sympathetic nerve branches are the carriers and conductors of these sensations of pain, is an unproved hypothesis.

The criteria enabling us to differentiate neuritis from neuralgia are the following. In the former the pain is generally continuous, though it may exacerbate at times; in neuralgia it occurs paroxysmally. In the first the nerve is sensitive to pressure in its whole course or for a long stretch; in the latter we find only pressure-points, or there is an entire absence of sensitiveness to pressure. In neuritis there is at times swelling of the nerves, which is absent in neuralgia. In the former symptoms of paralysis, atrophy, and anesthesia soon follow the pains; in neuralgia these are absent, or develop only in the latter stage, never, however, reaching a high intensity. Neuritis is generally an acute, neuralgia more often a chronic disorder.

In addition the following points are important for *differential diagnosis.* A diagnosis of neuralgia should not be made unless a close examination has excluded an organic disease, which may have elicited the pain by producing organic alterations in the nerves or central organs. Tabes dorsalis, tumor medullæ spinalis or cerebri, meningitis and syphilitic neuritis, tumors in the course or neighborhood of the affected nerves, aneurisms, etc., must be thought of. Neuralgic symptoms occur sometimes in multiple sclerosis also; I saw a case in which a severe trigeminal neuralgia was one of the first symptoms. On the other hand, neuralgia should not be diagnosed too quickly in *hysteric* individuals. Although a true neuralgia can develop in an hysteric individual, it is generally a *pseudoneuralgia*, pains which are either *purely psychical* or are due to peripheral irritation, which is so slight that only a mind altered by disease would react to them. These are *psychalgias.* These can be recognized by the fact that every psychical influence is able to influence the pain, to increase it temporarily, to silence, quiet, or evoke it, according to the conceptions and effect which it produces. It can often be noticed that the pain is under the influence of *self-observation,* increases under autosuggestion, and disappears when the attention is distracted. These pseudoneuralgias do not follow so strictly the nerve-paths; they occur in islets, diffusely or in *segments,* and are accompanied by corresponding symptoms of hysteria. The differential diagnosis is made more difficult by the fact that a true neuralgia can excite reflexly, in predisposed individuals, a hemianesthesia of an *hysteric* type, though this is rare.

The conception of neuralgia has been extended and has been referred to pains which appear at certain bony prominences or parts of joints without there being any noticeable local alteration. These conditions are generally of an hysteric nature. Other factors may, however, be in play. Remak and Bernhardt described a pain seated in the lateral epicondyles of the humerus, particularly of the right side, which came on especially after movements of the hand and fingers, and also after pressure. This *epicondylalgia*—probably caused by over-exertion of the muscles arising from this bony part—has nothing to do with neuralgia. Perhaps it should be grouped among the craft neuroses. It is possible that periostitic irritation helps to produce the pain (Remak).

Course and Prognosis.—Neuralgia may commence acutely, last some weeks, and then disappear forever. This course is not exactly the common one. More often it lasts for months and years; may, however, have remissions of longer duration. In some cases the attacks of pain occur at rare intervals and last only a short time, while the free intervals extend over a longer period. In other cases the attacks rapidly succeed one another, and the intervals between them are limited to some hours or days.

The prognosis is comparatively favorable in recent, acute commencing neuralgias, in youthful individuals, and in good constitutional conditions. The neuralgias following acute infectious diseases are generally less stubborn. The outlook is worse in persons of weakened constitutions, in conditions of exhaustion, in the senile, in the chronic intoxications, when the trouble has existed a long time, and when the pains are very severe. A neuropathic diathesis clouds the prognosis of typical neuralgia.

Treatment.—A thorough examination and exploration is a necessary preliminary to the treatment. If defective nutrition, loss of strength, or poor blood is the cause, a *betterment of the general condition* may suffice to silence the trouble. Consumption of an excessive amount of meat or undue use of alcoholic liquids and spices may be the exciting factor; passing from a rich albuminous diet to a vegetable one has been noticed to produce neuralgia. This circumstance must be kept in view. The diet should be a *mixed* one, not irritating; the condition of nutrition must be looked after; in some of my cases, recovery occurred after a *careful fattening*, in other, particularly stubborn cases, which had resisted all remedies, after a *forced diet* (*mastkur*). *Cod-liver oil* is praised for emaciated persons.

It is needless to urge the importance of a careful regulation of the diet in cases of diabetic and arthritic neuralgia, though abrupt changes and too much one-sidedness should be avoided.

The predisposition to neuralgic attacks may be combated by frequent cold washings and by a mild cold-water cure. Hydriatic treatment has also been recommended against neuralgia itself by Winternitz and Buxbaum; they praise particularly the Scottish douche.

Regulation of the bowels is an important point in the treatment of neuralgia. Cold-water clysters, rhubarb, and castor-oil often are of more value than nervines, and occasionally effect cures in old cases. If these do not accomplish the purpose, a course of treatment at Kissingen, Marienbad, or Homburg may do good. For cases of senile neuralgia Tarasp is a dangerous place.

In individuals accustomed to a sedentary life, regulated out-door exercise, riding, gymnasium work, etc., may have a favorable influence. Where over-exertion has been the probable cause, absolute rest, even continued rest in bed, is indicated.

If the nerve is imbedded in a scar, or subjected to pressure by a tumor or callus, proper *surgical* treatment is in place.

Recent neuralgias are occasionally quickly relieved by a *diaphoretic treatment*. *Local blood-letting* may also do good in such cases.

Malaria-neuralgia is generally relieved by quinine, which—after the individual idiosyncrasy to the drug has been ascertained—should be given in proper dosage. Generally fifteen to thirty grains are necessary. Quinine is best taken one-half to one hour before an attack. Where it fails, arsenic may relieve. Eucalyptol may also be tried. In anemia, *iron preparations*, perhaps *iron* in combination with *arsenic*, should be used; providing, of course, that the digestive functions are not harmed. Liq. ferri albuminati, Blaud's pills, hematogen, etc., are preparations that can be recommended.

If syphilis has preceded the neuralgia, even in apparently idiopathic neuralgia, a *specific* treatment, particularly potassium iodide, is indicated.

If intoxication with metallic poisons is the cause, *baths* (particularly sulphur baths), a *diaphoretic* treatment, *purging*, in combination with a strengthening diet, are curative factors. The most important measure is naturally the prevention of further intoxication, by removal of the injurious substance, change of occupation, etc. The indifferent baths and the carbonated mud baths are also at times of benefit, even in neuralgias from other causes, particularly in rheumatic cases.

To combat the neuralgia, *anodyne ointments* of opium, belladonna (1 : 10), cocaine (1 : 20–30 of vaseline), chloroform (with ol. hyoscyami and ol. olivarum, equal parts), and others are used. *Counter-irritants* and *revulsives*, either veratrin (0.5 : 20), aconitin (0.5 : 20), or *cantharides* or the *cautery*, are more efficacious, and may have a good influence even in old cases. In mild and recent cases moderate counter-irritation, as with cantharides, over the painful spots, is often sufficient; in old and severe cases the cautery is the best remedy. One or a number of superficial eschars will suffice.

In some cases cold has a good influence. Momentary cooling of the skin with applications of chloride of methyl or ethyl chloride (Debove, Reddard) may have a palliative effect.

Electricity is of much service. The galvanic current is particularly efficacious, especially the *stabile anodal treatment.* The positive pole is placed upon the diseased nerve, its points of exit, or upon a pressure-point, and the negative pole upon an indifferent location. It is advisable to commence in every case with weak currents, with 0.5–2.0 milliamperes with an electrode cross-section of about ten square centimetres, and if this does not suffice, increase the current strength. The current should be controlled by a rheostat; a sudden breaking is never permissible. Each sitting should last from one to five minutes, though a longer duration of single treatments to thirty minutes has been recommended. The faradic current can likewise be used, particularly the *faradic brush,* which evokes counter-irritation. Place the brush directly upon the places of exit of the nerve, or upon the pressure-points, or use a double brush or two brushes, which should be placed directly upon the nerves, the current being increased to as much as the patient can stand, even to 0. If pressure-points are found upon the vertebral column, it is advisable to bring these also under the influence of the anode.

I have personally seen good results in recent cases under the galvanic anodal treatment, recovery having ensued after ten to twenty sittings. This treatment was beneficial in several chronic and old cases, though it generally failed. In several which were particularly stubborn a *cataphoretic,* cocaine, or chloroform treatment, with a diffusion electrode, was crowned with success.

For this treatment, electrodes fitted with a space to contain the drug are necessary. This electrode is fitted to the positive pole. It is placed firmly upon the nerves, and a weak current of from one-half to two milliamperes is used.

If this treatment fails, *static electricity* can be tried; the sparks and breeze are recommended. In true neuralgia, however, I have not seen any success with this treatment.

The use of long-continued but very weak galvanic currents has been efficacious in some cases. The same is true of the electrical bath.

Massage is beneficial in sciatica, but does little good in trigeminal neuralgia.

I have no personal experience with the manual manipulations as recommended by Naegeli.

Climatic treatment can be recommended in many instances, although great successes are not to be expected from it. This includes a sojourn at the sea-shore, in the mountains, and in warm, equable climates.

Drugs are prescribed in order to cure the trouble, or to relieve the pains, or to silence them by producing narcosis.

Of the drugs which have been found useful in neuralgia, the following are the most important:

Quinine.—Commence with small doses, and increase to from thirty to sixty grains a day, if the smaller ones do not succeed and the drug is well borne.

Arsenic can be used in the form of Fowler's solution, from four to six drops as a dose, or in the form of arsenious acid, prescribed in pill form. An arsenical water (Levico, Roncegno) can also be used.

Sodium salicylate.—From forty-five to ninety grains a day in watery solution.

Salol.—In the same dose. *Salipyrin,* from five to fifteen grains.

The bromides. Potassium iodide, from five to ten grains several times daily.

Ol. terebinthinæ.—From five to fifteen drops in gelatin capsules.

Tr. gelsemii.—From fifteen to twenty drops several times daily.

Aconitin nitr.—.0001 (solution of 0.05 : 25.0, one drop, ten times daily, increasing to eight drops). *Cannabin. tann.,* from three to five grains.

Of the newer remedies: *antipyrin,* from five to fifteen grains; *phenacetin,* from five to fifteen grains; *lactophenin,* in the same dose; *antifebrin,* from three to ten grains; *analgen,* fifteen grains; *exalgin,* two or three grains in alcoholic solution; *methylene blue,* from three-twentieths to one grain (.01–.08) subcutaneously, or one and one-half grains (0.1) internally (highest daily dose, fifteen grains), in gelatin capsules; *butylchloral* (7.5, glycerin 20.0, aq. destill. 130.0, every ten minutes a teaspoonful), etc.

None of these drugs are reliable; their use has always only the value of an empiric attempt, and most of them require careful observation to prevent intoxication. Lately, *pyramidon,* from three to ten grains, has been recommended. I have several times seen good results from this drug in severe chronic cases. I have had no experience with *kryofin.* A very complicated treatment, in which strychnine, potassium iodide, and other drugs, in addition to rest in bed, were used, has been described by Dana.

Continued use of arsenic in small doses should always be tried in stubborn cases. In severe cases all these drugs fail, and the severity of the pains makes the use of *morphine* necessary. Subcutaneous injections in the vicinity of the diseased nerve are most efficacious, and to morphine is ascribed not only pain-stilling but also curative effects in neuralgia. Be careful not to place the hypodermic into the hands of the patient or relatives, and avoid a too frequent repetition of the injection. When morphine does not act satisfactorily, a combination of morphine and atropine has been praised. Cocaine may also be tried.

The use of *ether* and *methylene bichlorate* in the form of a spray has been advised, although in true neuralgia much cannot be expected from it.

Hypodermic injections of *perosmic acid* (one or two drops to a syringeful of a one per cent. solution) and carbolic acid have been used, but much cannot be expected of them. Lately, good results have again been claimed for osmic acid. These drugs should, if possible, be brought directly in contact with the diseased nerves.

The *ultimum refugium* of the treatment of neuralgia is *operative procedure;* at least, it should be. Krause warns us of hesitating too long; the knife should be used before the patient is driven into the morphine-habit. Bruns goes further, advising that severe cases should be immediately handed over to the surgeon, but he goes entirely too far. Simple section of the nerve—neurotomy—has generally only a transient result. This procedure has, therefore, fallen into disuse. Much more is accomplished by *neurectomy,*—by cutting out a large section of a nerve. *Nerve-stretching*, first used by Billroth and Nussbaum, has produced a cure in some cases. All these measures are, however, uncertain, and remissions can be expected in the majority of cases.

Nerve-extraction or exairesis, recommended by Thiersch and Witzel, in which the nerve is fastened by a special clamp and by screwing it up pulled out with all its branches, has produced good results, though remissions are not excluded (Angerer).

In neuralgia of the trigeminus, it has been decided resect to the branches of the nerve at the base of the brain, and lately even to extirpate the Gasserian ganglion (W. Rose, Horsley, F. Krause, Hartley). This operation has been successfully performed in numerous cases and has proven of great benefit. According to F. Krause's figures, of fifty-one patients operated upon five died, while the others not only survived the operation but were cured.

Ligature of the carotid has been tried, but can hardly be recommended.

Finally, it should be remembered that it has been attempted to cure neuralgias of spinal nerves by transection of the corresponding sensory roots within the vertebral canal (Bennet, Abbé, Chipault, Demoulin, Horsley). Nothing definite as to the indications for and the value of this operation can be ascertained from the reports at present.

NEURALGIA OF THE TRIGEMINAL NERVE (TIC DOULOUREUX, PROSOPALGIA, ETC.).

The trigeminal nerve is affected in neuralgia more often than any other nerve. In seven hundred and seventeen cases, collected by Conrads, it was implicated two hundred and thirty-nine times. This may be due to the extensive innervation of the nerve, to its exposed seat, and to its course through numerous narrow, bony canals. The factors given before as etiologic moments refer to *quintus* neuralgia also. This is especially true of a *neuropathic diathesis* which is in many cases the only cause discoverable. Malaria-neuralgia and neuralgia following upon the *infectious* diseases (particularly influenza) favor the trigeminal region, particularly the first branch. The same is true of the toxic forms. Whether there is a rheumatic neuralgia of the trigeminus, as, for instance, Leube describes, or whether cold acts only as a predisposing cause, cannot be definitely stated. Acute articular rheumatism is said to be able to clothe itself in the form of a neuralgia.

The disease is often due to diseased processes in the mouth, in the nose and frontal sinuses, particularly from carious teeth, abnormal formations of the teeth, exostoses on the same or on the alveolar process, abnormal condition of the teeth-pulps (Boennecken), chronic catarrhs, swelling and hypertrophy of the *nasal mucous membrane*, and *catarrh* of the *frontal sinus*. I have several times seen this disorder develop after operations within the naso-pharynx. Swelling of the periosteum within the bony canals through which the branches of the fifth nerve pass, with dilatation and inflammatory degenerative processes in the blood-vessels accompanying them, is often the cause. Moos found an exostosis upon the posterior wall of the external meatus to be the cause of a neuralgia of the fifth nerve.

The neuralgia which not rarely arises in the alveolar processes of a toothless jaw (*névralgie des édentés*) is referred to an otitic process in the alveoli, through which the nerve-terminals are irritated. According to Jarse, extraction of a tooth may elicit neuralgia.

Ocular disease (conjunctivitis, iritis, glaucoma, refractive errors) can likewise evoke neuralgia. It is more rarely due to a middle-ear catarrh. In some of my cases, continued *sojourn in overheated rooms* was ascribed as the cause.

Several times an aneurism of the internal carotid was the factor (Romberg).

Neuralgia of the fifth nerve is rarely bilateral (except in diabetes), and only exceptionally implicates all three trunks, but affects as a rule one or two or only a branch of these. A neuralgia originally confined to a small area may extend to several branches in its further course, or the opposite occurs; it can also spring over to other nerves, though this is not common. The pains are generally *very intense*, so pronounced that robust men style them unbearable. They can be compared to the entrance of a glowing hot rod or the boring of a sharp knife. The patient presses the hand to his brow, avoids all movement of the facial muscles, and is completely under the dominion of the pains. Their intensity is, however, subject to individual and periodic variation. At the height of a paroxysm, the pain may extend to the other trunks and even to other nerve paths (irradiation). The flashes of pain may come on conjointly, or steadily repeat themselves during several minutes or even hours. At the same time, *watering* of the eye of the affected side, occasionally also *increased nasal secretion* and *salivation* occur. *Twitchings of the facial musculature*, more rarely of the masticatory muscles, *reddening of the face, edematous swelling*, chemosis, even hemorrhage (for instance, of the gums) have been observed at the height of an attack. A diffuse swelling of the skin, the soft tissues, and the periosteum can likewise develop if the attack is long continued. Accommodation-paresis, difficulty in hearing, concentration of the visual field, taste hallucinations, and psychic disturbances (Laquer) are some of the rarer complications of neuralgia.

Herpes has often been observed, particularly herpes frontalis; it can also implicate the conjunctiva and result in inflammatory or even destructive processes in the eye. It rarely extends to the mucous membrane of the mouth and tongue. The cases in which neuroparalytic ophthalmia occurs in the course of the disease cannot be regarded as cases of true neuralgia. The changes in the color of the hair described are relatively frequent in facial neuralgia. Occasionally facial hemiatrophy comes on.

Rather often—according to Bernhardt, in two-thirds of all cases—the *first trunk* is affected (neuralgia ophthalmica), and here again most often the *supraorbital* nerve (*supraorbital neuralgia*). The pain is above the eyes, extends along the nerve to the border of the hair, to the coronary suture, even to the mesocranium. The pressure-point is at the supraorbital foramen. Malarial neuralgia often limits itself to this nerve, and shows so typically an intermittency that it has been called *intermittens larvata*. Influenza-neuralgia likewise favors this nerve. According to Seeligmüller, supraorbital neuralgia, due to affections of the frontal sinus, are also characterized by a typical course. In many cases the whole area of the first trunk is implicated; the pain radiates to the eyes, eyelids, and nose; pressure-points are probably in such cases found on the upper eyelid, ocular canthi, and nose. In neuralgia of the first (occasionally also the third trunk) there is at times a pressure-point upon the *tuber parietale*.

There is also a neuralgia limited to the eye (*ciliary neuralgia*): severe pains occur in the eye or are referred back of the eye, accompanied by watering, reddening of the conjunctiva, and photophobia. They are observed especially after diseases of the conjunctiva and cornea, in glaucoma, after injuries and over-exertion of the eyes, and in cases with refractive errors.

Neuralgia of the second trunk (supramaxillary nerve) occurs most often in the *infraorbital nerve.* The exit of this nerve at the infraorbital foramen is its most constant pressure-point. The alveolar or superior dental nerve is not rarely involved alone. The pains are felt in the upper jaw, even deep in the antrum of Highmore. If the whole trunk is implicated, the pain extends to the brow, nose, upper lip, malar bone, and temporal region. Pressure-points are then generally found on the temples,—on the anterior border of the temporal muscle,—upon the malar bone, the places of exit of the malar ramus, upon the upper gum, etc. The nasopalatine branch is rarely involved.

Neuralgia of the third trunk, inframaxillary neuralgia, when all branches are affected, evokes pains in the lower jaw, tongue, in the chin and temporal region, and also in the ear. They generally confine themselves to the alveolar nerve. Pressure-points are found corresponding to the points of its entrance into the inferior maxillary canal and the place of exit of the mental nerve.

There are mild and severe cases of fifth nerve neuralgia, some of which recover in a few weeks, others which extend over many years, even over decades. We can also speak of acute and chronic forms of fifth nerve neuralgia. I have treated persons with this disease in whom the trouble commences every year, generally at a definite time, to disappear again after a certain time. Changes of weather seem to have some influence. In some cases the pains are so severe that, when morphine becomes inefficacious, and the operations to which the patients willingly consent prove of no avail, *suicide* is attempted by the patients. These severe neuralgias—called by Trousseau *névralgies epileptiformes*—occur particularly in the aged. They are generally accompanied by loss of strength, as the ingestion of food is reduced to a minimum on account of the exacerbations of pain during the act of mastication.

The pains appear spontaneously, but are evoked and increased by *movements,* particularly of the facial and masticatory muscles, occurring therefore during speaking and chewing.[1] One of my patients could not touch his nose with his handkerchief, another could not touch his moustache, without being plagued with severe pains. On the other hand, firm pressure often relieves the pain. Psychic excitements may also excite the pain.

The diagnosis is generally easily established. Simple toothache does not follow the nerve trunks, but passes from a diseased tooth or from a certain point in the jaw, the painful spots being found here and not at the places of exit of the nerve. Periostitis and diseases of the bones are accompanied by diffuse pains and sensitiveness in the entire region innervated by the affected nerve. It may be difficult to determine whether the neuralgia is true, idiopathic, or *symptomatic*. Sclerotic processes at the places of exit of the trigeminus, tumors of the same region, in the neighborhood of the Gasserian ganglion, aneurisms of the carotid, etc., may hide themselves for some time under the symptoms of neuralgia. In its further course, however, other signs almost always appear which reveal the true trouble,—pain within the cranium, vascular murmurs in aneurisms, symptoms of intracranial pressure, paralysis of cranial nerves in tumors, etc. Some cases, however, have occurred in which a tumor—as, for instance, a cholesteatoma upon the Gasserian ganglion or in its vicinity (Romberg, Schuch, Krause-Benda)—was found to be the cause of a pure neuralgia of the fifth. In one case a calcareous concrement, pressing upon the supraorbital nerve, produced the neuralgia. Recent observations have also detected alterations in the branches of the trigeminus or in the Gasserian ganglion in the simple neuralgia. Dana found an affection of the vasa nervorum, Putnam sclerotic processes in the nerves, Horsley, Rose, Saenger, and Putnam sclerotic and degenerative alterations in the cells and fibres of the Gasserian ganglion. Krause rightly calls attention to the fact that the importance of these observations is doubtful, as they were found in individuals who had previously been subjected to manifold operative measures. Vascular alterations are certainly not the cause of neuralgia, but perhaps a result, as, from our experience (and the observations of Thoma, Debio, and others), arteriosclerosis may develop from long-continued vasomotor disturbances.

---

[1] In contrast, one of my patients claimed that chewing silenced the pain, so that he carried continuously a piece of fig-root in his mouth. In another case the neuralgia receded entirely during an attack of influenza, to reappear soon afterwards.

There is a form of neuralgia which is confined to the nerves of the scalp (*cranio-neuralgia*) and, as I have observed several times, may be combined with *alopecia*.

Otalgia (tympanic neuralgia) is, on the whole, little understood. The neuralgic pains in the ear occur in diseases of the pharyngeal-tubal mucous membrane, teeth, and maxillary joints. It can also develop from infection (B. Baginsky, Kaufmann).

Neuralgic pains without an anatomic basis occur also in the mastoid process (Schwartze), though it is questionable whether it should be regarded as neuralgia. In addition, the auricular vagus seems to be the seat of the pain.

Treatment.—The treatment should be preceded by a thorough examination of the teeth, facial and jaw-bones, nose and eyes. Generally the patient has attempted before he comes into our hands to obtain relief from the pain by having comparatively sound teeth extracted. At any rate, it is advisable to remove diseased teeth if they are sensitive to pressure or percussion, and particularly when by touching or shaking them a paroxysm is produced. In one case of neuralgia of the trigeminus which was combined with spasms in the facial, masseter, and neck muscles, I observed all symptoms disappear after extraction of a carious tooth.

If the teeth are healthy, exostoses or periostitic processes upon the maxilla may be the cause. Neuralgia of toothless individuals is said to have disappeared in a few cases after resection of the alveolar processes.

Chronic coryza, swelling of the nasal mucous membrane, should be treated locally. Two of my patients, however, asserted that they only began to be troubled with severe neuralgia after their noses had been examined or treated with instruments.

In a neuralgia arising in the frontal sinuses, *nasal douches* (with warm water, or weak salt, calcium chloride, or boric acid solution, perhaps with the addition of cocaine) are recommended.

Errors of refraction should be corrected by appropriate glasses.

Occasionally it is possible, as the experience of Gussenbauer has shown, to relieve the neuralgia by the administration of purgatives and cold-water clysters.

In addition, what has been said in general under the caption of neuralgia applies also to trigeminal neuralgia. Electricity is of much value. Whenever direct (galvanic, faradic, static, cataphoric) treatment fails, galvanization directly through the cranium (corresponding to the position of the Gasserian ganglion) or of the cervical sympathetic may be tried. Occasionally a pressure-point is found on the vertebral column, on the upper cervical vertebra, and in such cases the application of the anode to this place may be advantageous. Prolonged galvanic currents—for a whole hour—have produced good results in several cases. V. Frankl-Hochwart has used the double-brush electrode with benefit in these cases especially. Electrotherapy, however, often fails.

As to drugs, I would particularly advise the use of sodium salicylate, quinine, arsenic, tr. gelsemii, and of potassium iodide. Cocaine instillations into the conjunctival sac or painting the nasal mucous membrane with a five to ten per cent. cocaine solution may act as anodynes. Butylchloral is said to have occasionally acted favorably in small doses (0.1–0.2, from one and a half to three grains). Inhalation of amyl nitrite (from two to four drops upon a handkerchief) during an attack is said to have sometimes cut it short. Aconitine, narceine, napellin, neurodin, extr. stramonii, bromidia (one or two teaspoonfuls), and others are recommended. Among the newer remedies, *pyramidon* deserves to be particularly praised. Schleich recommends subcutaneous local applications of a solution of 0.1 (one and a half grains) cocaine, 0.02 (about one-quarter grain) morphine, 0.3 (four and a half grains) sodium chloride, and 100.0 (about three ounces) water.

In some cases, in which nearly everything had been tried, I undertook an energetic irritation of the soles of the feet with the faradic brush, and produced temporary good results.

Trousseau used quinine and opium in full dosage (even eight or ten grammes a day) in stubborn cases.

Compression of the carotid may shorten the paroxysm. The use of nerve-vibrators, massage of the cervical sympathetic (Rossander), Naegel's manipulations, etc., are not of much benefit.

Finally, neuralgia of the fifth is the best field for *operative* treatment. Where other measures have failed, surgical treatment is in place. This includes *neurectomy*, which more often evokes remissions than recovery, *extraction* of the nerve, after Thiersch, which does much good according to many surgeons (only Kraus is somewhat sceptical towards it), and, finally, *intracranial resection* of the nerve or of the *Gasserian ganglion*, and of the trunk of the fifth according to Krause and others. This method and its results and dangers have already been spoken of. The writings of Vogt, Schede, Angerer, and particularly the monograph of F. Krause, describe the methods used in these various operations.

OCCIPITAL NEURALGIA.

Of the upper four cervical nerves the major occipital nerve is most often involved alone, though occipital neuralgia is far rarer than trigeminal neuralgia. In fifteen thousand cases of neuralgia there are, according to Remak, about fifty in which the occipital nerve is the one affected. The other branches, the minor occipital, auricul. magnus, subcut. colli, and the supraclavicular, are implicated still more rarely. I have treated two cases of this kind in which the neuralgia affected the whole region innervated by these nerves, and in which pressure-points were not only found at the places of exit of the major occipital—between the mastoid process and the upper cervical vertebra in the middle—but also upon the posterior border of the sternocleidomastoid, just where the nerves surround it.

The pain generally confines itself to the region innervated by the major occipital nerve; it is, however, more often bilateral than unilateral. It ascends, therefore, from the nuchal region over the posterior part of the head to the vertex. The pains are generally very severe, not always intermittent, but at times continuous and exacerbating in impulses. Every movement of the head, laughing, coughing, sneezing, and rapid walking increase it. The head is held stiffly backward and inclined to the side.

The most constant pressure-point is the one which represents the place of exit of the major occipital nerve.

Hyperesthesia of the skin of the back of the head, loss of the hair in this region, and swelling of the cervical glands are accessory symptoms. Seeligmüller also gives: contraction of the pupil and reddening of the ear in the attack upon the side most affected, tinnitus aurium, and gastric disturbances.

Sneezing, watering of the eyes, etc., are also said to occur occasionally during an attack, symptoms which are explained by the anatomic relations of the occipital and trigeminal nerves. A combination of neuralgia with symptoms of involvement of the cervical sympathetic was described by Johnson. This can be referred to a disease-process in the upper cervical ganglion of the sympathetic.

In one case I found painful spots (tubercula dolorosa) upon the cervical nerves.

Among the *causes* of this form of neuralgia, the following are the most important: carrying of heavy weights upon the head, trauma and colds, infectious diseases (malaria, typhus, cerebrospinal meningitis, influenza), gout, and arthritis deformans. The neuralgia occurring in the last-named disease is probably always a *symptomatic* one (compression of the nerves from vertebral disease). The occipital pains which come on in caries of the upper cervical vertebræ, should not be included here. They may correspond exactly to an occipital neuralgia, but are to be referred to a traumatic neuritis. The strict avoidance of all movements in this disease, the sensitiveness of the vertebræ, the swelling in the neighborhood (tubercular granulations, abscess), the occasional crepitation present, the anesthesia of the occipital region which comes on later, and finally symptoms denoting involvement of the spinal cord, the spinal roots, and the veins arising in the medulla oblongata, generally allow a correct diagnosis to be established.

In rheumatism of the cervical and nuchal muscles, the muscles themselves are sensitive to pressure, the pain has a diffuse extension, increases generally in bed, extends to the shoulder muscles, etc.

Hysteric nuchal pain most often causes error. Hysteric individuals complain often of pains in the nape of the neck. Generally, however, they tell you that the pain is not only in the nuchal region but extends from the spinal cord, even from the small of the back upward and over the head to the eyelashes. The pressure-points are also not confined to the places of exit of the major occipital nerve. Hyperesthesia of the skin of the head and nucha is very marked. The psychogenous origin of the pain is almost always detectable through some suggestive influence. Finally, other signs of hysteria are generally present.

The prognosis of pure occipital neuralgia is, on the whole, good, though very stubborn cases occur, as, for example, an observation of Jastrovitz teaches.

Treatment.—In recent cases local inunctions of anodyne ointments, moderate bloodletting, a hot or a Priessnitz's pack, or diaphoresis are in place. A vapor bath may do immediate good.

Galvanic electrization can also be recommended (particularly a stabile anode treatment), the efficacy of which has lately been again praised by E. Remak. If weak currents do not do any good, occasionally a surprising result can be secured by the use of very strong currents, which are to be conducted through the upper cervical vertebræ, or, by placing the electrodes directly upon the occipital pressure-points, are carried directly to the nerves.

Irritative ointments, cantharides, local applications of tincture of iodine, and, in particularly stubborn cases, the actual cautery, can also be tried.

Quinine acts well, especially in malarial (intermittens larvata) forms, but sometimes, also, in ordinary neuralgia. In addition, consult the general chapter on neuralgia.

NEURALGIA OF THE PHRENIC NERVE.

Little that is reliable is known of this form. The pains are said to follow the course of the nerve: from the diaphragm through the breast (between the pericardium and pleura) up to the neck, and are combined with a feeling of oppression. Pressure-points are found upon the points of insertion of the diaphragm, as well as on the nerve itself, where it is electrically stimulated.

During the paroxysm the respiration is generally difficult and painful.

This neuralgia is most often found accompanying diseases of the heart, pericardium, and of the large blood-vessels. When such diseases are not present, the prognosis is said to be favorable. In one case which I saw, neuralgic pains were present in the region innervated by the phrenic after a fracture of the clavicle. In another case which I saw, it seemed to be a case of intermittens larvata; the attacks occurred only at night.

A bilateral form of this neuralgia has likewise been described. The diagnosis cannot be established with absolute certainty.

NEURALGIA BRACHIALIS.

Neuralgia of the brachial plexus involves the region innervated by the four lower cervical and the first dorsal nerves or a part of it. Only rarely does it limit itself strictly to the course of the brachial nerve, though, as a rule, one—for example, the musculospiral or the ulnar—is most severely implicated. More often an indefinite, not sharply localizable, extension of the pains is present, which also occasionally radiate to the upper intercostal nerves, nerves of the shoulder, and even to the trigeminus. Either they occur in paroxysms or they are to a slight degree continuous, increasing from time to time in severity. They are described as boring, burning, lightning-like, etc. (causalgia). The patient instinctively grasps his arm and seeks to place it in a position in which every pressure and pulling can be avoided. Some amelioration is secured by supporting it with the sound hand or carrying it in a sling. As movement seems to increase the pain or to excite the paroxysms, muscular action in any form is repressed.

Pressure-points are often found on the affected nerves, particularly on the musculospiral, where it winds around the upper arm, on the ulnar (between the internal condyle of the humerus and the olecranon), on the median in the elbow or over the wrist, and on the circumflex nerve where it gives off its cutaneous branch. The cutaneous branches may be sensitive to pressure where they pass through the fasciæ. Pressure evokes not only pain but also paresthesias in the regions innervated by these nerves, so that the patients sometimes can describe rather well the anatomic course and branching of the nerves in the peripheral parts. Occasionally, pressure-points are found over the spinous processes of the lower cervical vertebræ or beside them. The rarer cases, in which the pains occur only during certain movements (piano-playing, manual labor, enamelling, etc.), should be separated from the neuralgias and regarded as a form of craft neurosis.

Paresthesias (formication), pallor and flushing of the skin, hyperidrosis, and rarely herpes, are *concomitant phenomena* occurring in neuralgia. The individuals generally complain of a feeling of heaviness and rigidity in the arms. Severe *trophic* disturbances, observed particularly after shot-wounds of the nerves, probably always denote a neuritis, and do not justify a diagnosis of neuralgia, though it is difficult to draw a line between them. Hyperesthesia of the skin is often present, in the later stages probably also hypesthesia. A marked decrease of sensation is, however, rare in pure neuralgia; it indicates a neuritis or a central lesion.

A *neuropathic* diathesis is an important causative factor in brachial neuralgia. Most cases are neurasthenic or hysteric. In *hysteria* not only vague pains of uncertain localization occur in the arms, radiating almost always to the neighboring regions of the trunk and head, combined with paresthesias and hemihyperesthesia, but in some cases a pseudoneuralgia closely simulating the brachial form.

Anemia and *cachexia* also tend to predispose to this disease. In two of my cases diabetes was the cause, and in one the neuralgia disappeared with the sugar in the urine. *Traumata* are particularly often blamed, and it is just the most severe forms of nerve-pains which may be of traumatic origin. A neuritis is probably produced in most cases either from direct injury of the nerve-trunk or by irritation from a strange body, a scar, a splinter of bone, or a callus. In former times venesection gave rise occasionally to a neuralgia resulting from a nerve lesion. In those cases in which an injury of the peripheral nerve branches of the fingers (compression, bite, and so forth) is the cause of the trouble, we have generally a reflex neuralgia, which can be differentiated from an ascending neuritis. The *traumatic reflex neuralgias* are, however, often only part of the symptoms of a general traumatic neurosis.

Brachial neuralgia may be due to *rheumatic* influences. Malaria, typhoid, and influenza sometimes cause it.

Diseases of the heart and aneurisms of the aorta and subclavian are occasionally accompanied by neuralgic troubles in the arm, especially the left, while in diseases of the liver the pain is in the right arm. In one case in which a diagnosis of brachial neuralgia had been made I found an aneurismatic dilatation of the subclavian; the musculospiral area particularly was involved, and atrophy of the triceps showed that neuritic or degenerative alterations had taken place in the nerve. The same cause was observed in a few other cases. The upper ribs through pressure may also evoke this form of neuralgia.

Differential Diagnosis.—One must be particularly cautious not to confuse this disease with diseases of the spinal cord and its roots. Pachymeningitis cervicalis hypertrophica, vertebral caries, tumors arising in the meninges, etc., can for a long time hide themselves under the phenomena of a brachial neuralgia. The symptoms, however, in such cases occur generally in both arms; only *tumors* cause often a unilateral neuralgia. The further course, however, always reveals the fact that a destructive malady is present as paralysis, atrophy, and anesthesia come on. The pressure-points along the nerves also are generally absent, while the spinal column is the seat of spontaneous pains, and some vertebræ are very sensitive to percussion. In cervical tabes the pains likewise occur on both sides, and other characteristic signs are present. In muscular rheumatism the pain does not follow certain nerve tracts; not the nerves but the muscles themselves are sensitive to pressure, particularly their places of insertion, and the pain is not of a neuralgic character.

All in all, I regard a true brachial neuralgia as a rare affection; generally hysteria or an organic disease or a constitutional disorder (diabetes and the like) is back of it. Since the appearance of the first edition of this book I have been enabled to enlarge considerably my experience in this respect, and have become more and more convinced that brachial neuralgia is generally a *brachialgia* or a psychalgia brachii,—*i.e.*, a pain in the arm of indefinite localization which is the symptom of a neuropathic or psychopathic general condition, and which generally occurs in conjunction with other nervous manifestations, particularly depression, insomnia, irritability, etc. The brachialgia often masks the neurosis or psychosis. I have observed these conditions far more frequently in males than in females. Generally the detection of its psychogenous origin is not difficult.

The remarks made in the general consideration of neuralgia hold true here likewise in regards to **prognosis** and **treatment.** In recent cases, try diaphoresis and revulsives, the latter also in advanced cases. The application of flying blisters over the nerve-trunks, the use of a cautery, etc., are particularly advisable. Quinine, arsenic, sodium salicylate, oil of turpentine, phenacetin, pyramidon, etc., should be tried in order. Belladonna is also included among the curative drugs. Electrical treatment often does good.

After what has been said above, it can readily be seen that treatment of the psychogenous origin of the malady is essential. The chapters on hysteria and neurasthenia should be consulted for this.

In stubborn cases *nerve-stretching* can accomplish much. Other surgical measures (extirpation of tumors) may be in place. Section of the corresponding spinal roots is permissible only in severe cases which have resisted all other curative measures

Nägeli described and recommended a non-bloody method of nerve-stretching. In a case of aneurismatic dilatation of the subclavian artery with arteriosclerosis, in which neuralgia, later compression-neuritis, had been diagnosed, recovery ensued under the simultaneous use of potassium iodide, electricity, and local application of an ice-bag.

INTERCOSTAL NEURALGIA.

The neuralgia which affects the nerves arising from the dorsal roots affects almost entirely or especially the anterior branches,—the intercostal nerves. Those of the left side are more often implicated. It rarely confines itself to one, but generally extends over the region innervated by several neighboring intercostal nerves. Occasionally the pain extends to the inner side of the arm. The pains, as a rule continuous, are, however, subject to pronounced exacerbations, and in some cases become very severe; they often occur only in the anterior and lateral parts of the thorax. They follow the course of the intercostal nerves, and may also localize themselves in a circumscribed area. Some of my patients experienced, in addition to the pains running along the intercostal spaces, stitch pains passing from before backward through the breast. They came on spontaneously, and were increased by concussions, coughing, sneezing, and deep inspiration.

The *pressure-points* are of diagnostic importance. There are generally three, one close to the spinal column at the height of the origin of the diseased intercostal nerves (vertebral point), one in the axillary line corresponding to the ramus perforans lateralis (lateral point), and one beside the anterior median line of the body where the rami perforantes anteriores appear, also upon the sternum and rectus abdominis. It is uncommon to find the intercostal nerve sensitive to pressure in its whole extent.

The skin corresponding to the innervation of the affected intercostal nerves is occasionally hyperesthetic; even the slightest touch or pressure of the clothes suffices to evoke pain. Anesthesia is rare, and its presence renders it improbable that we are dealing with a pure neuralgia; hyperesthesia, however, occurs. Intercostal neuralgia is just the form which is most often accompanied by *herpes*, though the relations are not constant, and it is probable that the neuralgia which is accompanied by herpes zoster is always a *neuritis* (Curschmann and Eisenlohr, Dubler), or that a material disease of the spinal ganglion (v. Bärensprung and others) or of the spinal roots is the cause. The neuralgia may precede the development of the herpes and last much longer.

The pain may radiate to the back or to the arm or shoulder of the same side. Intercostal neuralgia sometimes is combined with angina pectoris. In one of my patients a bilateral intercostal neuralgia came on regularly during the night, so that he was awakened by the pain. Lues was not present.

This form of neuralgia affects particularly women of youthful or mature age, but often occurs in men and the senile.

Hysteric individuals complain often of intercostal and side pains. They may be true neuralgic, but more often are hysteric pseudoneuralgias. Formerly it was thought that intercostal neuralgia was more frequent upon the left side, and especially in the region supplied by the intercostal nerves (the fifth to the ninth), on account of circulatory disturbances, and particularly to difficulty in the flow of blood from the veins in this area on account of the anatomic relations (Henle). It is probable that the cause of this is to be found in the relation of intercostal neuralgia to hysteria in which the sensory anomalies favor the left side. Self-observation directed to the heart is probably also a predisposing factor to left-sided intercostal neuralgia. It may also be combined with disturbances of the cardiac innervation (*brachycardia, tachycardia*), though it also occurs that these phenomena as well as the neuralgia are co-ordinated symptoms of a cardiac disease.

Anemia, cachexia, and conditions of exhaustion following upon lactation, the puerperium, pyrexial diseases, and loss of blood form a favorable soil for this disease. Sexual excess, onanism, diseases of the uterus and of the ovaries are likewise of etiological importance. A bilateral intercostal neuralgia of syphilitic origin with nightly exacerbating pains has been described. Injury to the ribs or fracture of the ribs may be the exciting cause leading to the development of a neuralgia; generally, however, a traumatic neuritis resulting from a direct nerve lesion, or compression of the nerve by a callus, is the cause. *Scoliosis,* as also all deformities of the spinal column, may favor the onset of intercostal neuralgia.

The intercostal pain occurring in diseases of the vertebræ and spinal cord is indeed generally of a neuralgic character, but should be separated from intercostal neuralgia.

We know nothing certain concerning the genesis of that form of this malady which occurs occasionally in diseases of the lungs and pleura; it is perhaps in most cases a tubercular neuritis. Aneurisms of the aorta may cause neuralgic pains by direct compression of the intercostal nerves. These may also occur reflexly in the manner described by Head (page 59).

The prognosis is doubtful. The disease is generally of long duration without endangering life, providing it is not a symptomatic form. A close examination of the vertebral column, the cardiac and vascular apparatus, the lungs, and of the functions of the spinal cord prevents any confusion with symptomatic neuralgia. Tumors of the spinal cord particularly may, however, evoke nothing but symptoms of a true neuralgia for years. The differentiation of neuralgia from rheumatism of the thoracic muscles and pleurodynia is not difficult.

Treatment.—Whenever a cause can be discovered, the treatment must be directed to it. Conditions of exhaustion and constitutional defects must be particularly combated. If the pain can be referred to a callus, a poorly set fracture, a tumor on the ribs, etc., the process causing the irritation must be removed. If a chronic distortion of the vertebral column is present, the proper orthopedic treatment must be instituted. If a badly compensated heart failure is found, the use of digitalis may favorably influence the neuralgia, as I observed in two cases. In addition, the measures advised above for the treatment of neuralgia should be consulted. Subcutaneous injections of osmic acid (one per cent.), hydrochloric acid (two per cent.), and ether have been recommended.

Operative treatment is proper in very severe cases. Nerve-stretching has often cured the neuralgia. Schede had a good result with resection in one case. I saw one case in which galvanic electrization was successful after stretching of the nerve had failed; another, in which several of the intercostal nerves were resected for a large extent without any benefit being secured.

Concerning section of the spinal roots, see page 351.

NEURALGIA OF THE MAMMARY GLANDS; MASTODYNIA; IRRITABLE BREAST (ASTLEY-COOPER).

There is a form of neuralgia limited to the nerves of the mammary glands. These nerves arise from the intercostal nerves, the skin over the mammæ being supplied by the lateral and anterior perforating rami of the second to the sixth intercostal nerves and the gland itself by the lateral perforating rami of the fourth to the sixth intercostal nerves.

The pains occur in paroxysms, and may reach a high degree. They are generally combined with *hyperesthesia* of the skin, particularly with excessive sensitiveness of the nipples. Circumscribed *reddening* and *swelling*, even a *local swelling* of the glandular substance, often simulating a tumor, can occur. Erb and Lesser have observed the excretion of a milk-like fluid during or after the attacks. In isolated cases vomiting occurs at the height of the attack. Herpes is rarely seen in conjunction with this form of neuralgia.

Pressure-points are generally observed over the second to the sixth spinous processes of the vertebral column, and not upon the nerves of the mammary glands. *Tubercula dolorosa* are sometimes noticed upon the mammary nerves.

The malady affects women of mature age almost entirely. It has been observed only a few times in men. Anemia and hysteria are often causative factors. Pregnancy, lactation, fissures of the mammæ, traumata, and mental excitement may evoke the neuralgia. During menstruation the pains generally exacerbate.

The diagnosis is not difficult to establish. Only where a circumscribed hardness is found in the glandular substance is a differentiation from malignant tumor difficult. In neuralgia, however, such intumescences are not constant, do not show any gradual increase in size, and do not lead to swelling of the lymphatic glands.

The prognosis as to recovery is not favorable, as this neuralgia is very stubborn and but slightly amenable to treatment.

Treatment.—Duchenne recommended the use of the faradic brush. The constant current may also act well, as I observed in one case. Binding up the breasts lessens the pain. A slight pressure bandage, warm clothing, anodyne ointments, etc., may also still the pain. The coating of the nipple with a strong cocaine solution has occasionally a palliative effect. Nothing can be added to what has been previously said in regard to drugs. Nägeli recommended a kind of stretching of the mammæ.

NEURALGIA LUMBALIS.

Lumbar neuralgias have been divided into those of the short and those of the long lumbar nerves. The first are those which supply the lumbar, perineal, hypogastric, inguinal, and a part of the genital region, —*i.e.*, the ileohypogastric, the ileo-inguinal, and the genitocrural with its two branches, the external spermatic and the lumbo-inguinal. Neuralgia of these nerves is also called *lumbo-abdominal neuralgia*. The long lumbar nerves are the anterolateral femoral cutaneous, the crural, and the obturator. Each of these can become involved in neuralgia, the crural being the most frequently implicated.

Lumbo-abdominal neuralgia is characterized by pains, which radiate from the lumbar region to the perineal and lower abdominal region, into the inguinal crease, scrotum, and perhaps into the spermatic cord. The upper part of the anterior surface of the thigh may also be affected. Now this, now that branch is most involved. Pressure-points are found beside the lumbar vertebræ, above the crests of the ilium, beside the linea alba, on the inguinal canal, scrotum, etc. Cutaneous hyperesthesia is occasionally present; herpes zoster is also not a rare concomitant symptom. A cramp-like contraction of the abdominal muscles, cremasteric spasms, and vomiting are also occasionally observed at the height of a paroxysm. As the pains increase in walking, the patients avoid walking altogether, or walk slowly and carefully with small steps. Sexual excitement, even ejaculation of semen, and increased flow of urine are given as rarer symptoms.

Crural neuralgia may confine itself to the anterolateral femoral cutaneous, the pain being then upon the outer surface of the thigh as far down as the knee-joint, a pressure-point being found upon the anterior superior spine of the ilium. I saw this neuralgia develop twice after influenza, once after gonorrhea and acute articular rheumatism. (See also Meralgia Paræsthetica, page 276.)

If the *crural nerve* is involved, the pain follows the course of this nerve and its branches, particularly the anteromedian femoral cutaneous and internus, and also the saphenus major, and passes down the inner surface of the leg to the inner border of the foot. A pressure-point is found over the crural nerve in the inguinal crease, occasionally also in the course of the saphenus major. The pains are increased by movements of the legs, particularly by extension of the thigh. *Vasomotor* disturbances, *hyperidrosis*, and *herpes* are also not rare in this type of neuralgia. Paresthesias and particularly anesthesia render it probable that an organic disease is the cause.

The obturator is most rarely involved. The pains run along the obturator nerve from the obturator foramen upon the median surface of the thigh down to the region of the knee.

All these forms of neuralgia must be diagnosed with caution. In most cases it is of a *symptomatic* form. Tumors on the lumbar vertebræ and in the pelvis particularly can be hidden for a long time under a lumbo-abdominal or a crural neuralgia. Only the most exact examination, which consists not only in palpation of the abdominal walls, but also per anum and per vaginam, can prevent gross errors in diagnosis. Among the causes of the idiopathic forms, cold, over-exertion, traumata, obstipation, masturbation, and diseases of the sexual sphere are given. Diabetic neuralgia not rarely localizes itself in the region supplied by the crural and obturator nerves, though neuritis is generally the cause. (See page 275; see also the account of the so-called intermittent limping in the next chapter.) The neuralgia may follow diseases of the scrotum or of the urethra, whether from reflex action or through a neuritis ascendens is not known. Injuries of the ankle-joint or distortions of the tibiotarsal joint occasionally evoke a neuralgia of the saphenus major.

The obturator neuralgia is generally due to pressure and laceration of the nerve in obturator hernia.

The prognosis of idiopathic lumbar neuralgia is not unfavorable. Recovery can be expected particularly in young and strong individuals. The general part of this work and the next chapter must be consulted for the treatment.

NEURALGIA ISCHIADICA; SCIATICA (MALUM COTUNNII).

Sciatica is that form of neuralgia which affects the sciatic nerve and its branches, and sometimes the cutaneous femoris nerve, and occasionally extends to other roots of the lumbosacral plexus.

The disease is very common. Men are affected much oftener than women, particularly those of middle age. It is not rare in the old, while children are almost exempt.

It must be acknowledged that, if it is difficult to draw a sharp line between neuralgia and neuritis, it is particularly the case for this form. There is no doubt that the symptoms of sciatica are often due to a *slight neuritis* of the sciatic, which occasionally reaches a sufficient intensity to expose through clinical signs its true character. In most cases these signs are missing, but it is firmly established that *every stage of transition between neuralgia and typical neuritis occurs.* For this reason, in discussing the etiology we cannot make a differentiation, but must include as causes of sciatica many factors which, according to our conceptions, produce a mechanical lesion of the nerve and inflammation of it.

A *neuropathic condition* increases the predisposition to this disease, but it is not so important here as in the other neuralgias. Sciatica affects rather often persons who until then were well, the strong and fat as often as the lean. It may develop after *gout* (Gowers, Hyde,—according to this author, this is one of the most frequent causes) and after *diabetes mellitus* (Worms, Ziemssen). Bilateral sciatica is particularly often of diabetic origin. The relationship to *syphilis* is less certain, if one excludes the fact that gummata have been observed in the nerves. Chronic *intoxication* (alcohol, lead and other metallic poisons) may also be the cause. It occasionally occurs after the acute infectious diseases; it may even follow gonorrhea (Fournier). There is no doubt that in some cases it results from a *muscular rheumatism* of the lumbar muscles; true *myositic* processes can cause an inflammation in the neighborhood of the sciatic and thus be a cause.

Exposure to cold is a prominent cause of sciatica. Soon after a cold —particular danger exists in sitting upon a cold and wet support— it is seen to develop. *Traumata* are also of importance. Excluding direct injuries of the nerves, we may include here *contusion* of the perineal region, falling upon the hips or buttocks, etc. Sitting upon a hard support, even, can produce sciatica in lean individuals. Overexertion of the legs through the stress of occupation is also blamed (Seeligmüller).

If the sciatic or its roots are subjected to compression, this is often first expressed by signs of sciatica: pelvic tumors, gravid uterus, the head of the child, etc., can act in this manner. The *mechanical* lesions to which the sacral plexus is subjected in *childbirth*, particularly the use of forceps, may produce sciatica. Even the pressure influence of hard masses of feces has been given as a cause.

It is doubtful whether *venous stasis* in the pelvis, in the venous plexus accompanying the sacral nerves, can produce a sciatica. Inflammatory processes in the pelvis, particularly *perimetritis*, may affect the sciatic region.

The disease is generally unilateral. *Bilateral* sciatica is, as a rule, symptomatic,—*i.e.*, caused by diseases of the spinal cord, by tumors of the vertebral column, sacrum, and pelvis, or by constitutional affections (particularly diabetes); an idiopathic sciatica may also, however, involve both sides.

Symptoms.—The symptoms are not, as a rule, very severe at first, but increase in the course of some days or weeks. In the beginning the patient has only a disagreeable drawing sensation on the posterior surface of the thigh, or exclusively in the gluteal and lumbar region, which soon, however, becomes a pronounced piercing, drawing, boring, or even a lightning-like pain, which generally passes through the extremity from above downward. These pains follow the course of the sciatic nerve, beginning in the gluteal region, where it emerges from the sciatic foramen, or higher up, pass, corresponding about to the middle line of the thigh, down the posterior surface, and are transmitted to the peroneal nerve and its branches or to the tibialis posticus.

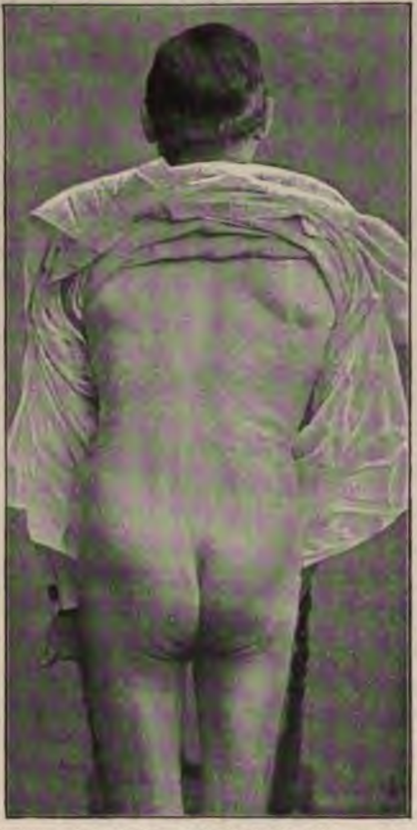
They can pass in this way to all parts of the leg and foot except the inner part supplied by the crural nerve or the saphenus major. They generally follow only one nerve, so that *the patient may follow the tract of the pain with his finger.* The posterior femoral cutaneous is often affected; sometimes the neuralgia is confined to this area. Only in a few cases have the plantar nerves alone been involved. This plantar neuralgia may cause very severe pains (Romberg).

The pains are located in the skin or deeper. They either appear in paroxysms or exacerbate paroxysmally, particularly at night.

They are evoked and increased by pressure, movements, and an uncomfortable position, and walking is therefore difficult. The patients seek to relieve the sick limb, supporting themselves as much as possible upon the sound limb, particularly avoiding movements which lead to a stretching of the sciatic,—*i.e.*, flexion of the thigh with extended knee or extension of the leg with flexed hip. As a rule, the leg is held a little flexed at the hip- and knee-joints, while the floor is but slightly touched by the sole of the foot.

According to Albert, Nicoladoni, and others, a scoliosis of the lumbar vertebral column (ischias scoliotica) is not rare, the concavity being towards the sound side. (Fig. 178.) The occurrence of this crossed scoliosis has been ascribed to various factors. It has been assumed that it is a result of the endeavor to relieve the sick limb, and that for this reason the centre of gravity is shifted to the sound side. Others (Schüdel, Kocher, Gussenbauer) believed that the sacrolumbar of the diseased side is stretched and extended in order to protect a sensory branch which is involved in the sciatica, or that the patient instinctively widens the intervertebral foramina, thereby lessening the pressure upon the emerging roots (Nicoladoni). These views are opposed to that of Mann, who regards the scoliosis as being due to a paresis of the erector trunci of the diseased side.

FIG. 178.



Patient with sciatica of the left side and secondary scoliosis.

A scoliosis towards the diseased side (homologous scoliosis, according to Brissaud) also occurs, which has been referred to a reflex spasm of the erector trunci. Contracture of this muscle can occasionally be distinctly detected, and should not be confused with a simple bulging of the muscle-belly, which in crossed scoliosis is due to torsion of the spinal column. Remak described an alternating scoliosis. A kyphosis develops much more rarely.

Walking increases the pain, though occasionally the opposite has been noticed. In sitting also the patient assumes a peculiar position, resting on the tuber ischii of his sound side to guard the diseased sciatic from pressure. Protracted sitting and reclining are, as a rule, not well borne. Coughing, straining, and sneezing excite pain, and are, therefore, repressed as much as possible. . The sciatic nerve is only rarely painful to pressure in its whole extent, but generally a number of *pressure-points* are found. The most constant ones are the following: one beside the superior posterior iliac spine, over the place of exit of the nerves from the major sacrosciatic foramen; one at the lower border of the gluteus maximus, between the trochanter and the tuber ischii; one in the middle of the popliteal space; another below the small head of the fibula; often also malleolar pressure-points, etc.

At times the sensitiveness of the sacral nerves may be detected per anum or per vaginam. In some cases pressure-points are absent. The sensitiveness of the nerves may be demonstrated in another way. The patient is asked to lie down and his leg is then flexed upon the hip-joint. Generally pain is not felt on the posterior surface of the thigh or in the gluteal region when the leg is raised a foot or so, sometimes only after it forms an angle of from ninety to one hundred degrees with the pelvis. This pain disappears as soon as the leg is flexed upon the thigh; it is, therefore, due to stretching of the sciatic nerve. This *sciatic phenomenon* (Laségue) is of great diagnostic importance. It can also be elicited in a sitting posture, by bringing the thigh into an extreme position of extension.

Sensory disturbances are generally absent; occasionally there is a slight hyperesthesia in the region of the tibialis posticus or peroneus. The tendon-reflexes are generally increased in the diseased limb, occasionally on the sound side also, though the Achilles-tendon reflex may be lost (Sternberg, Babinsky). In some cases I have noticed a flaccidity and abnormal movability of the Achilles tendon.

Slight weakness in the flexors of the leg is occasionally noticed. Here and there *fibrillary tremor* is observed, and, in chronic cases, a slight atrophy. If qualitative alterations in electrical excitability (partial reaction of degeneration) are found, it is a case of *neuritis* and not of neuralgia. Vasomotor disturbances are rare, as is also herpes. Coolness of the skin occurring in spots has been mentioned by Erben as a frequent phenomenon. At the height of a paroxysm the pain may radiate to other nerve-tracts, even into those of the lumbar plexus; muscular contractions and even clonic spasms of the legs may come on. Symptoms pointing to an extension of the process to the plexus have also been observed, as, for example, paresis of the gluteus maximus. In a few cases glycosuria has been noticed (Braun, Schiff). Polyuria is also said to occur (Debove).

I wish to call attention to a complication which occurs in the course of sciatica, not alone in hysterical women, but also in men who previously were healthy,—*i.e.*, a reflex neurosis, characterized by weakness, tremor, paresthesia, and sensory disorders in the half of the body corresponding to the seat of the sciatica.

Differential Diagnosis.—Sciatica is too frequently diagnosed. Many physicians call every pain which is limited to a leg, sciatica. Its characteristic signs, the pain along the tract of the sciatica, the pressure-points, the pain on stretching the nerve, the disorders of motility resulting therefrom, etc., have already been described. The negative signs must also be emphasized—absence of paralysis, of marked anesthesia, of degenerative atrophy, etc.

A marked anesthesia, especially one accompanied with atrophy and a reaction of degeneration, as has been described by Nonne, Guinon, Parmentier, and Charcot, indicates neuritis, and must never be confounded with neuralgia. Absence of the Achilles-tendon reflex on the side affected speaks also for neuritis, though I would not exclude sciatica from absence of this sign alone.

The pains accompanying spinal diseases almost never limit themselves to the sciatic nerve of one side. Other symptoms in these cases show the nature of the disease, as paralysis of the lower limbs, bladder weakness, etc.

Diseases of the *hip-joint* produce pains in the hip- and knee-joints which do not follow the nerve-tracts; the movements of the joint are painful, especially the driving of the head of the femur into the acetabulum.

See the chapter on hysteria for the symptoms of *nervous coxalgia.* In muscular rheumatism the pains spread diffusely; the pressure-points are absent, and squeezing of the muscles and pressure upon their insertions produce pain. *Hyperesthesia plantaris* should not be confused with sciatica. It is due to neuritis of the plantar nerves (hysteria, gout, alcoholism).

Individuals with hemorrhoids may have pains of an indefinite nature and extent in the legs. They are probably due to irritation of the sensory nerves in the periphery, perhaps also to venous dilatation in the vertebral canal and pressure of the varices upon the emerging roots within the vertebral or sacral foramina. In sciatic pains caused by compression of the nerves, the pressure-points are said to be absent; signs of dissolution of continuity also occur later. A careful examination of the pelvis (per vaginam and per rectum) must be made.

Hysteric sciatica is characterized by pains of an indefinite localization and character, accompanied by psychical alterations.

Intermittent limping (or claudication) was first noticed in horses (Bouley). Charcot described it as seen in men. It consists in a feeling of numbness, pain, and fatigue, occurring in one leg on attempts to walk, and increased by exertion, so that locomotion may be hindered, and later (perhaps after one-fourth of an hour) may be impossible. After a little rest the trouble disappears. We do not notice anything objectively which indicates a nervous trouble; on the contrary, *arteriosclerosis* was generally found, and the weak, or absent, pulsation of the arteries of the extremity involved was especially noticed (Charcot, Bieganski, Goldflam).

There is no doubt that the disease, as Charcot showed, is due to a narrowing of the lumen of the large arteries (iliac, crural) from arteriosclerosis or arteritis obliterans, causing defective nutrition of the muscles.

Marinesco found pronounced alterations in the musculature as a result of obliterating arteritis. The malady is often a prodrome of gangrene. It occasionally occurs in conjunction with diabetes. It is not known whether an arterial spasm due to vasomotor disturbance can cause intermittent limping. Neither can we exclude the question whether arteriosclerosis is capable in itself of producing local pain (Nothnagel, Laache, Lang).

Achillodynia (Albert, Schüller, etc.) should not present any differential diagnostic difficulties. It consists of severe pains which arise at the point of insertion of the Achilles tendon in walking and standing. Occasionally a swelling is also found : it is probably a bursitis. It is said to ensue after gonorrhea, malaria, and traumata. Similar troubles occur in the region of the unciform bone (talalgia, tarsalgia), partly from local disease processes, partly without any objective signs.

A myositis of the calf-muscles occurring particularly after over-exertion may also give rise to a false diagnosis.

The nature of *Morton's metatarsalgia*, a severe pain in the region of the fourth metatarsophalangeal joint, is still obscure. A gouty diathesis, tight shoes, exercising pressure upon the nerves of the foot,—that is, upon the periarticular nerve branches,—over-exertion from continued standing, general nervousness, etc., have been given as causes. It has also been thought to be a true neuralgia of the external plantar nerve. The condition rarely occurs in the other joints of the toes.

Course and Prognosis.—It may end in recovery in a few weeks or months, or it may become chronic and last years, with remissions and exacerbations, and after recovery leave a predisposition for a recurrent attack. Rheumatic and slight traumatic cases have the best prognosis. The general condition has much to do in the matter. The prognosis is unfavorable in old cases, in the senile, and when due to a basal disease which cannot be relieved.

Treatment.—Rest and disuse of the parts are indicated. A comfortable position should be secured. Hard seats are to be avoided, etc. In recent cases a diaphoretic treatment is advisable : a sweat-bath with wet packs following it. *Bloodletting* over the chief pressure-points may do much good (leech, scarifier). Cold is generally not well borne, while hot cloths often ameliorate the pains. The local spraying of chloride of methyl or of ethyl chloride, as well as the external application of condensed carbonic acid, has been recommended. Revulsives are often efficacious, particularly flying vesicants, which do good in old cases also; large Spanish-fly blisters over the pressure-points, put on simultaneously, or one after the other. Superficial scarification with a cautery over the sciatic nerve, particularly at places corresponding to the pressure-points, is often efficacious. It is not advisable to repress the suppuration too long. Cauterization of distant places, even the lobes of the ear, has been done several times with good results. An old but not to be discarded remedy is the emplastrum oxycroceum, which produces an eczema.

If the diaphoretic treatment fails, one can immdiately use electricity, but only after previous attempts at revulsion have failed. To the electrical treatment is ascribed a pronounced therapeutic action by almost all neurologists. The most efficacious method of treatment is the galvanic. A large electrode is placed upon the nerve corresponding to its place of exit, the other upon one of the peripheral pressure-points, in the hock of the knee, the calf of the leg, etc. The current should be of average strength, though some favor weak and others strong currents. It is advisable to commence with a weak current and to vary the dosage according to the effect observed. The treatment can be altered by gradually shoving the electrodes over the nerve so that a part of it is constantly directly under the influence of the electricity. It is also rational to place one electrode upon the gluteus maximus over the greater sacrosciatic foramen, the other *vis-à-vis* in the inguinal region, and to send a strong current through them.

The faradic brush is not more efficacious than any other form of peripheral irritation, though it permits daily renewal of the irritation and does not cause any unpleasant symptoms. Very strong currents are necessary. In old cases, energetic faradization of the muscles is said to have been beneficial several times. Bernhardt and others prefer static electricity. The continued wearing of a galvanic element might also be mentioned.

Massage does much good. It recommends itself especially in chronic cases. It would also often be beneficial in recent cases, if "force" cures were not attempted and the irritated nerve still further maltreated. It is advisable to begin with weak rubbings and kneadings, and to confine them, if possible, to the neighborhood of the nerve. In rheumatic and gouty forms, massage has often an excellent effect. Treatment by compression of the pressure-points, particularly the upper one upon the sciatic notch, is recommended by Negro, and Arullani has constructed a special apparatus for the use of this method.

In respect to drug treatment, salicylate of sodium, oil of turpentine (in capsules, 1.0 each, three to ten daily), arsenic, quinine, antipyrin, and phenacetin are the chief ones used; in many cases (even where syphilis cannot be demonstrated) *iodide of potassium* acts efficaciously.

To combat the severe pains we use the *narcotics*, though it is advisable to abstain as much as possible from the use of morphine injections. If one must do it, the hypodermic needle should be inserted at the site of the pain. Nitroglycerin (one to five drops of a one per cent. solution) is praised by Krauss.

If obstipation exists, if hard fecal masses are present in the intestines, *purgatives* are advisable; a large dose of castor oil may relieve the patient in an almost miraculous manner, though a single evacuation is generally not sufficient. The obstipation must be continually counteracted by proper laxatives or by a water-cure.

It may be necessary to replace the uterus, to remove a tumor, to prescribe an anti-arthritic diet, to order a forced diet (*mastkur*), etc. Bruns saw one case with lateral flexion of the uterus recover immediately after its reposition.

Old cases are sometimes cured by a stay at one of the *indifferent springs*, by treatment at Teplitz, Gastein, Wiesbaden, Wildbad, Warmbrunn, Baden-Baden, and Ragaz. Mud baths and the hot sand baths (of Köstritz) must also be mentioned. *Hydrotherapy*, particularly the use of the Scottish douche, has also been recommended. As a last resort, we have *nerve-stretching*, which must naturally be avoided when the signs of a neuritis are pronounced. It may be efficacious if the nerve is bound down by a connective-tissue cicatrix. Some authors have had good results with bloodless extension of the sciatic. Bonuzzi's method (page 138) may be used for this purpose. The scoliosis does not need any special treatment; with the recovery from the sciatica, the deformity disappears, as I have repeatedly observed.

In Morton's metatarsalgia absolute rest and avoidance of all tight foot gear is necessary; a particular form of shoe (Gibney), and finally, in severe cases, operative removal of the metatarsal head, have been recommended.

PUDENDOHEMORRHOIDAL NEURALGIA.

Neuralgia of the pudendohemorrhoidal plexus is rare but stubborn. It occurs most often in the testicular region as *spermatic neuralgia*. It is characterized by severe pains radiating from the region involved to the inguinal region, passing along the spermatic cord to the scrotum and epididymis, or from them along the spermatic cord to the inguinal region. The skin of this region becomes very sensitive (irritable testis). During the paroxysm, spastic contractions of the cremasteric, twitchings of the legs, vomiting, priapism, ejaculations, even periodic swelling of the testicles, are some of the symptoms that may occur. This disease is very stubborn to treatment, and produces considerable depression, and has even led to suicide. It is not definitely known whether the neuralgia has its seat in the external spermatic nerve or in the branches of the sympathetic.

The use of a suspensory, electrical treatment, and antineuralgic drugs are the methods adopted for cure. Forcible compression of the spermatic cord has been tried. In some cases castration was decided upon. In one case resection of the scrotal nerves led to recovery.

It must not be confounded with tumors (tubercular, etc.). The lancinating pains of tabes particularly involve the testicles, and in many cases it is a psychical disease. An *anoperineal neuralgia* (W. Mitchell) has been observed in onanists, and also sometimes in individuals who do not masturbate. In one case I secured a cure by means of cocaine suppositories. In another all remedies failed, and the patient finally became a morphomaniac and wandered from one institution to another.

Under the name of *rectal neuralgia* have been described pains which occur at every bowel evacuation and last for a long time. Cases of this kind must be very rare. I have seen some persons who complained of an extraordinary weakness following each defecation. Neuralgias of the bladder, urethra, prostate, and penis have also been described.

COCCYGODYNIA.

This refers to a severe neuralgic pain in the coccygeal region, which occurs almost entirely in women. The pain occurs spontaneously in or from sitting, walking, and evacuations of the bladder or rectum. The coccyx is sensitive to touch and to movements. It may occur after trauma or from difficult labor, or in hysteria. In many cases an inflammatory process is at the root of the trouble. A careful bimanual examination should, however, enable us to distinguish such conditions from neuralgia. There are slight cases in which the use of opium suppositories relieved the condition in a few days or weeks; others in which a psychrophore was of benefit, and severe cases in which nothing relieved the patient and an operation was necessary (Simpson, Koenig, and others). Electrical treatment has been known to cure many cases. Seeligmüller claims to have cured a coccygodynia of twelve years' standing by faradization (an electrode in the vagina and one upon the coccyx). Others report similar experiences. Where hysteria is a factor psychotherapy is demanded.

VISCERAL NEURALGIA.

Severe pains in the internal organs of a neuralgic character are not rarely observed. They accompany diseases of these organs or are due to some nervous disorder. For instance, neuralgic pains in the region innervated by the gastric nerves belong to the phenomena of gastric crises. *Gastralgia*—severe pains, coming on paroxysmally, in the epigastrium, radiating towards the back, combined at times with vomiting

—may belong to the symptoms of hysteria or form a hemicranial equivalent. A pure neuralgia of the gastric nerves independent of these basal diseases seems to occur in otherwise healthy individuals, though it is rather rare. To combat it, we use belladonna, cocaine, codeine, bismuth subnitrate, morphine, and silver nitrate.

A pure neuralgia of the intestinal, hepatic, splenic, and renal nerves has not been positively observed. These organs are, however, not rarely the seat of severe pains in the functional neuroses, particularly in hysteria. Gowers reports a case in which attacks of pain occurred in the region of the kidney for forty years, without renal calculi being found or even evidence of their former presence having been demonstrated.

According to Head, the peripheral pains accompanying abdominal disease, which generally correspond to an over-sensitiveness of a certain part of the skin, affect the area of innervation of the corresponding spinal cord segment; these areas of the skin derive their nerve-fibres from the same roots or spinal segment from which come the sympathetic nerves of the internal organs involved. Visceral pain is reflected along these peripheral nerves. In herpes zoster, he found that the same areas were affected.

NERVE TUMORS.

Various neoplasms may arise from the nerves: *neuroma*, *fibroma*, *sarcoma*, *glioma*, *myxoma*, and *syphiloma*. Carcinomatous and syphilitic infiltrations of peripheral nerves are probably always secondary to tumors of the neighboring nerves. Among these neoplasms, neuromata are of special interest. Virchow's classification into *true* and *false* neuroma, according as to whether the nervous tissue (medullated and non-medullated fibres) has or has not a considerable part in the structure of the tumor, has been accepted by many. Billroth, Ziegler, and others, however, have written in opposition to this separation; the latter, particularly, has doubted entirely the implication of the nerve-fibres in the neoplasm. True neoplasms, according to him, do not exist, but only *neurofibroma*, *neurosarcoma*, and *neuromyxoma* arising in the peri- and epineurium. Neurolipoma may also occur.

Neuromata may come on *singly* or have a *multiple* extension. They form on all nerves, but especially on the spinal. Multiple neuromata may confine themselves to one nerve-area,—for example, to the brachial plexus, the cauda equina, the pelvic nerves, etc.,—or extend over the nerves of the whole body. One thousand have been found in one person. Occasionally we find a rosary-like arrangement upon the nerves. (Fig. 179.) The circumference of a neuroma varies between that of a pea and a child's or man's head; they attain, on an average, the size of a pea, a nut, or a pigeon's egg.

Tubercula dolorosa is the name given to small tumors, the size of a milium to a pea, occurring singly or in large numbers on the sensory peripheral nerves, distinctly palpable and generally painful. They are probably always *fibroma*.

Plexiform neuroma (congenital elephantiasis) is a congenital tumor due to a cord-like thickening and plexus-like dissolution and combination of the fibres of a nerve. It occurs particularly on the trigeminal, but also in other nerves, is very rare and need not evoke any symptoms. The cords can be distinctly felt under the skin. The relations of this disease to pachydermatocele and elephantiasis mollis cannot be discussed here.

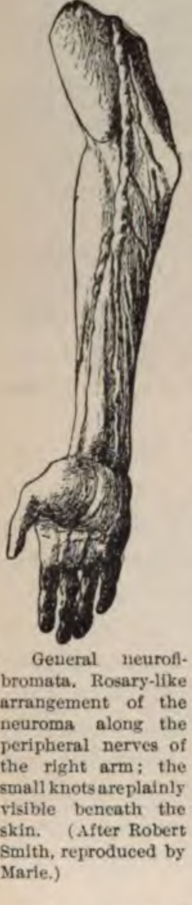
General neurofibromatosis (fibroma molluscum multiplex) is the name given to a condition in which numerous fibroma (molluscs) are found upon the branches of the peripheral nerves, also neuromata upon the nerve-trunks and pigment spots (nævi) upon the skin.

V. Recklinghausen has shown the relationship existing between the molluscs and the peripheral nerves.

Little that is reliable is known concerning the etiology of neuromata.

Multiple neuromata are, as a rule, of *congenital* origin. The *neuropathic constitution* is also an etiologic factor; the disease may be hereditary and familial, and still only develop in later life. A relationship to idiocy has also been shown. *Tuberculosis* is said to predispose to this malady. *Traumata* may be a direct cause of their formation or through the production of a chronic neuritis.

FIG. 179.



General neurofibromata. Rosary-like arrangement of the neuroma along the peripheral nerves of the right arm; the small knots are plainly visible beneath the skin. (After Robert Smith, reproduced by Marie.)

Symptoms either are entirely missing or correspond more or less to those of a *neuralgia* or a *neuritis*. Neuroma may remain latent and be discovered accidentally during an examination or only at the post-mortem. They are often confused with lipomata.

Even where the tumor forces apart the fibres of the nerve, all signs of a break in conduction may be absent. Generally these tumors expose themselves by the *severe pains* which they cause and which radiate towards the periphery from a particular spot. Marked *sensitiveness to pressure* is also present, and when the neoplasm is superficial in position, palpation will reveal an intumescence upon the nerve or a movable tumor.

If it arises from the nerve itself, it cannot be displaced along its longitudinal axis, in contradistinction to the paraneural tumors. As a rule, *paresthesia*, occasionally *hypesthesia*, occurs in the area supplied by the affected nerve, and rarely paralysis and atrophy. Reflexly, local *muscular contractions*, and even general convulsions of the type of reflex epilepsy, may develop. A combination of the disease with epilepsy I have noticed once; with hysteria, hypochondriasis, and neurasthenia several times.

The general condition remains undisturbed if the neoplasm causes but little or no pain. With severe neuralgic symptoms, marasmus may gradually develop. General neurofibromatosis often leads in its later stages to marasmus. Psychical disturbances, sensory disorders, and convulsions occur with this disease (Marie). Life itself is not directly endangered. Neuroma of the cauda equina may, however, cause paraplegia and paralysis of the bladder, etc., and death result through such symptoms. It should also be remembered that neuromata not rarely transform themselves into sarcomata.

The disease is *slowly progressive*; it may, however, come to a standstill, or even retrogress.

If the tumor is solitary and can be reached, its *extirpation* is indicated if it elicits pronounced symptoms. Where possible, the nerve stumps should be directly united, or by the use of a suture *par distance*, etc. (page 242). Several times, notwithstanding excision of a large section of the nerve, symptoms of degeneration were absent or receded very rapidly. The tumor is sometimes so slightly adherent to the nerve that it can be separated from it without its continuity being disturbed.

If it is a multiple tumor, it may still be advisable to cut out those which cause most of the trouble. In malignant cases, it is the rule for them to return. In general we must needs confine ourselves to a *symptomatic* treatment, to combating the pains. It should be noted that some electrotherapeutists (M. Meyer) report the destruction of these tumors by the use of *galvanic currents*, percutaneously applied.

SECTION III.

DISEASES OF THE BRAIN.

ANATOMY AND PHYSIOLOGY OF THE BRAIN. LOCALIZATION.

THE description of cerebral diseases will be preceded by a short discussion of the anatomy and physiology, grouping the facts which are necessary for the physician to know before he can enter the field of brain pathology.

THE MEMBRANES OF THE BRAIN.

The dura mater is at the same time the inner periosteum of the cranial bones. In adults it can easily be stripped from the inner surface of the cranium at most places; only upon the base of the cranium does it adhere more firmly to the bones.

Wherever the dura forms the sinuses, it divides into two membranes, also where nerves are imbedded in it. It forms further in the cranial cavity free processes. The large vertical process, the *processus falciformis major*, extends from the crista galli to the internal occipital protuberance, and is fastened to the lateral borders of the sagittal sulcus by two membranes. It dips in between the two hemispheres so that its lower border is only two millimetres from the upper surface of the corpus callosum. Above, the *superior longitudinal sinus* is surrounded by the two membranes and by the dura covering the sagittal sulcus, while the lower border surrounds the *inferior longitudinal sinus*. In addition to the superior longitudinal sinus, cavities (parasinoidal spaces) are found in the dura mater on both sides of the median line. Here the cranial veins enter before they reach the sinus. The *processus falciformis minor*, or the falx cerebelli, extends from the internal occipital protuberance to the foramen magnum, and lies between the two cerebellar hemispheres. The outer border adherent to the crista occipitalis forms the occipital sinus.

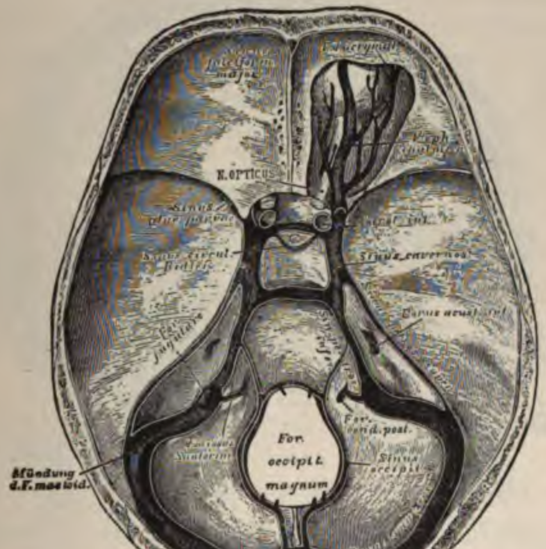
The transverse process, the *tentorium cerebelli*, lies between the lower surface of the occipital lobes and the upper surface of the cerebellum. Its insertion is at the lineæ transversæ of the occipital bone, where it forms a part of the *transverse sinus*, and at the upper angle of the petrous bone where it surrounds the *superior petrosal sinus*. The anterior free border is deeply indented (incisura tentorii). The median part of the tentorium lies upon the monticulus cerebelli.

Figs. 180 and 181 show the position and course of the sinuses, without any further explanation being necessary.

Processes of the dura mater, dural sheaths, accompany the emerging cranial nerves. The optic nerve is accompanied by a dural sheath as far as the eyeball; the sheaths of the acoustic and facial enter the internal auditory meatus with them, and extend even into the facial canal.

The arterial vessels arising from the middle meningeal artery are found upon the outer surface of the dura, and are seen on the inner surface of the cranium in the well-known fissures for the blood-vessels. The veins of the dura are connected with those of the cranium by numerous branches. The nerves of the dura are branches of the sympathetic fibres accompanying the arteries; the fifth nerve also sends a few branches (nervi spinosus and tentorium cerebelli) to the dura.

FIG. 180.



The blood-vessels of the dura mater. Bird's-eye view. (After Heitzmann.)

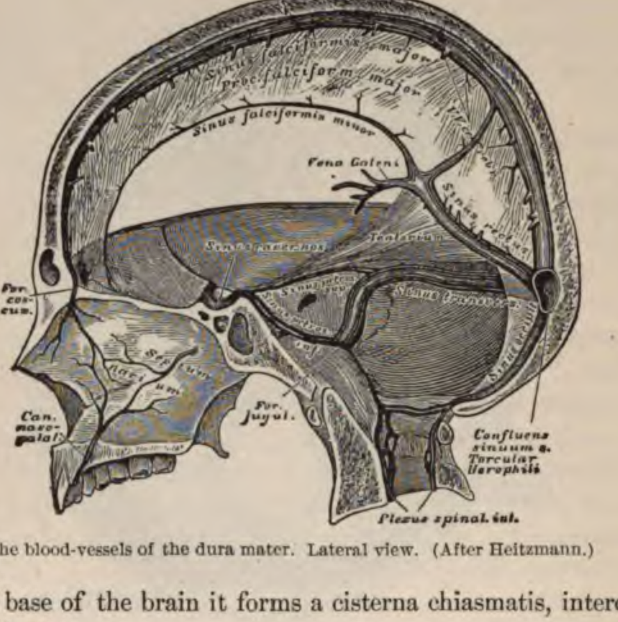
The small space between the dura mater and the arachnoid, the subdural space, contains but little fluid. The *cerebrospinal fluid* is found in the subarachnoidal space and in the ventricles.

The arachnoid is joined to the pia at many places by the processes and bands forming the subarachnoidal connective tissue. There is, therefore, properly speaking, no subarachnoidal space, but a number of smaller and larger intercommunicating subarachnoidal spaces.

The arachnoid and pia would form one membrane upon the convexity did not the first span itself over the fissures, and did not the pia, which lies everywhere close to the upper surface of the brain, cover the bottom of the fissure.

On the base of the brain the arachnoid is separated from the pia for great distances, so that we find here large subarachnoidal spaces (cisternæ subarachnoidales or subarachnoidal sinuses). The cisterna magna cerebellomedullaris is formed in this way, the arachnoid stretching from the dorsal surface of the medulla oblongata directly to the posterior part of the lower surface of the cerebellum as far as the superior vermis, while it does not enter the space between the inferior vermis and the tela choroidea. The medulla oblongata is surrounded *in toto* by a wide subarachnoidal space.

FIG. 181.



The blood-vessels of the dura mater. Lateral view. (After Heitzmann.)

On the base of the brain it forms a cisterna chiasmatis, intercruralis, fossæ Sylvii, etc.

Within the subarachnoidal space are found the larger cerebral blood-vessels.

The *Pacchionian bodies or granulations* (arachnoidal papillæ) are knotty, rapecole-like growths from the arachnoid, which generally force themselves, in the region of a dural sinus or a parasinoidal space, into the dural tissue, and thin this so much as to produce their well-known impressions upon the inner surface of the cranium. The papillæ may even grow through the bone. They are separated from the venous spaces themselves by a thin layer of the dura mater. The tissue composing these papillæ is a continuation of the subarachnoidal tissue. It has been shown experimentally that an excess of serous fluid may pass from the subarachnoidal space into the sinuses of the dura through these bodies when the pressure is slight in the sinuses. The arachnoid also forms sheaths for the nerve-roots.

The subarachnoidal spaces communicate with the cerebral ventricles. The communication is through the fourth ventricle at its hind end, where the *foramen Magendii* is found,—a rather large oval lacuna in front of the calamus scriptorius, and also through two lateral lacunæ corresponding to the tops of the recessus laterales.

The *pia mater* lies everywhere close to the upper surface of the brain, and as the *tela choroidea* dips into the ventricles. The tela choroidea superior, which passes transversely through the cerebrum, covers the third ventricle. The venous plexus accompanying it is continued through the foramen of Monro into the lateral ventricles. The internal cerebral veins are found in the tela choroidea superior. These unite at the posterior end of the pineal gland to form the *greater Galenic vein.*

The inferior tela choroidea lies between the ventral surface of the cerebellum and dorsal aspect of the medulla oblongata.

The pia contains sympathetic nerve-fibres, derived from the plexus surrounding the arteries of the circle of Willis; branches of some of the cranial nerves also enter it.

THE CONVOLUTIONS AND SULCI OF THE UPPER SURFACE OF THE BRAIN.

The positions and courses of the gyri and sulci can be seen by an inspection of Figs. 182, 183, and 184.

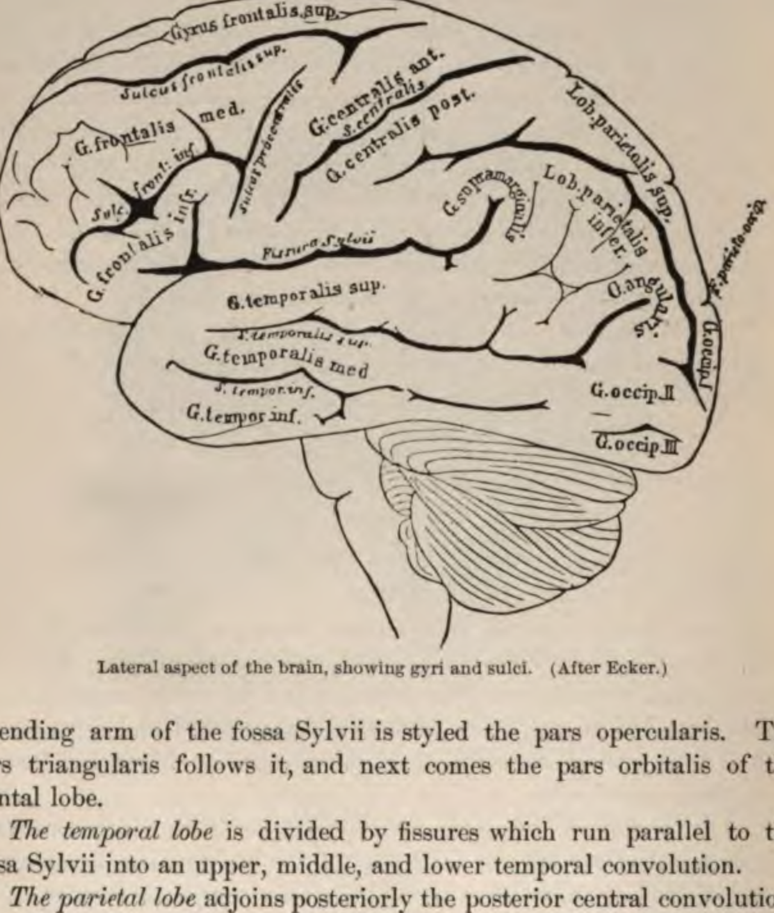
It is most easy to locate places upon the upper surface of the brain if one use the *fossa Sylvii* and *sulcus centralis* as guides. The fossa Sylvii, which consists of a long posterior and two short anterior arms, passes from before and below backward and upward and separates the frontal lobe, central convolutions, and a part of the lower parietal lobe from the temporal lobe.

At the bottom of the fossa lies the *island of Reil*, which consists of several small convolutions. The part of the brain which lies above the island of Reil is called the *operculum*. This comprises the foot of the central convolutions, the posterior part of the third frontal convolution, and the part of the lower parietal lobe lying between the interparietal sulcus and the fossa Sylvii.

In the operculum there arises a well-marked important fissure, which extends from below and front upward and back to the surface of the brain. This is the *sulcus centralis* or fissure of *Rolando.* It is bordered in front by the *anterior*, in back by the *posterior central convolution* (gyrus centralis anterior and posterior).

The frontal lobe is divided into three sagittal running convolutions, separated by two fissures. They are generally connected by transitional convolutions (transitional gyres). The lowest is called the third. The part of the third frontal convolution which is cut off by the anterior ascending arm of the fossa Sylvii is styled the pars opercularis. The pars triangularis follows it, and next comes the pars orbitalis of the frontal lobe.

FIG. 182.



Lateral aspect of the brain, showing gyri and sulci. (After Ecker.)

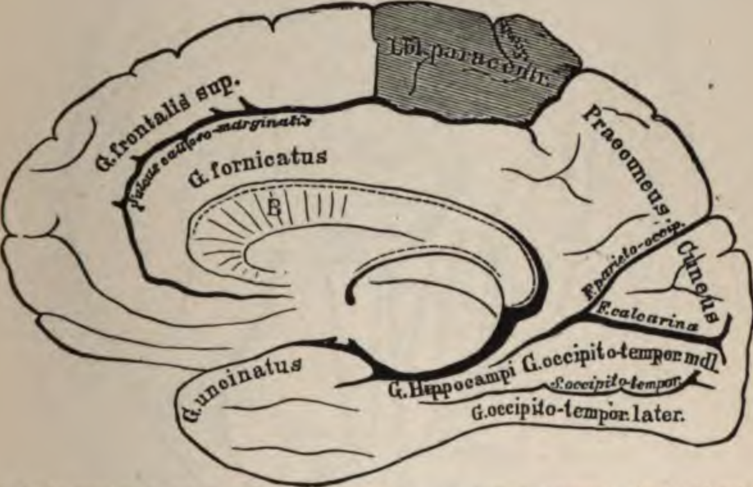
The temporal lobe is divided by fissures which run parallel to the fossa Sylvii into an upper, middle, and lower temporal convolution.

The parietal lobe adjoins posteriorly the posterior central convolution. It is divided into an upper and a lower parietal lobe by a fissure, *the interparietal sulcus,* which is arched and is often interrupted. The upper lobe soon passes into the posterior central convolution ; the lower consists of the *gyrus supramarginalis,* which surrounds the posterior end of the fossa Sylvii, and the *gyrus angularis*, which embraces the upper temporal fissure. This latter is continued dorsad into the posterior parietal gyre. The interparietal fissure can be divided into three sections, which are also occasionally separated from one another, the anterior one being called the fissura retrocentralis inferior and the posterior one the sulcus occipitalis anterior.

The occipital lobe is separated from the parietal lobe by the *anterior occipital fissure* and also by the *fissura parieto-occipitalis* on the median side.

On the median surface of the hemisphere wall (Fig. 183) we find just above the corpus callosum the *gyrus fornicatus*, whose upper border is formed by the *sulcus callosomarginalis*. It is continued behind by the *precuneus*, which represents the median surface or median area of the parietal lobe. In front of it lies the *paracentral lobe*, a direct continuation of the central convolution. The precuneus is bordered behind by the fissura parieto-occipitalis.

FIG. 183.

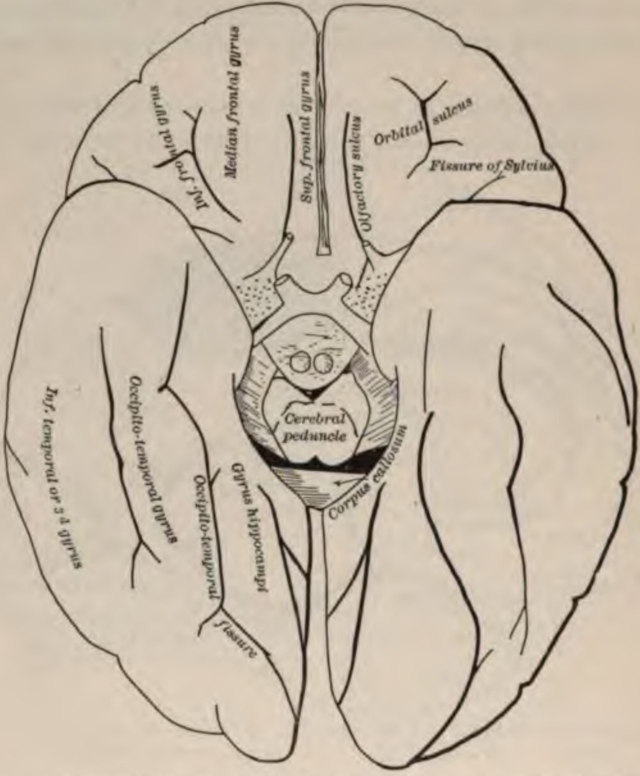


View of the median upper surface of the cerebrum. B, corpus callosum. Gyri and sulci are marked. Paracentral lobe is shaded. (After Ecker and Strümpell.)

A wedge-shaped area, the *cuneus*, is formed on the median hemisphere wall by the union of this fissure and the *calcarine fissure*.

The gyrus fornicatus (callosal gyre, according to some authors) runs in a curve around the *splenium* of the corpus callosum to form, as the *hippocampal gyre*, the upper convolution upon the median surface of the temporal lobe. It ends in its anterior part as the *gyrus uncinatus*. A convolution lying below the calcarine fissure, the *lobus lingualis*, passes in front directly into the hippocampal gyre. The gyrus fusiformis, which adjoins this, is separated by the sulcus temporalis inferior from the third temporal convolution. The convolutions at the base of the brain can easily be learned from Fig. 184.

FIG. 184.



The convolutions at the base of the brain. (From Edinger. Schematic after Ecker.)

THE RELATION OF THE CRANIUM TO THE SUPERIOR SURFACE OF THE BRAIN.

This is shown in Fig. 185.

The frontal bone covers most of the frontal lobe, the lower convolution completely.

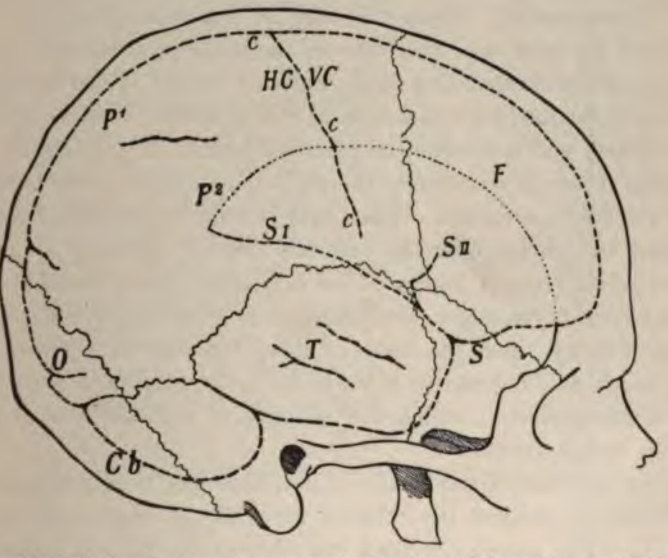
Below the parietal bone are the central convolutions, the parietal lobe, and a part of the occipital lobe. The parietal tubercle corresponds about to the lower parietal lobule, or to the supramarginal gyre. The temporal bone covers the greater part of the temporal lobe, the squamous sutures lying with the highest point of their arch over the fossa Sylvii;

from about one to one and a half centimetres above this point the sulcus Rolandi begins.

This has almost constant relations to the coronal suture, its lower terminus lying about twenty-eight, its upper arm from about forty-eight to fifty-five millimetres behind the coronary suture. As this suture cannot always be palpated, it is necessary to know other methods which allow the position of the convolutions and fissures to be ascertained without any attention being paid to the course of the cranial sutures.

To locate the upper end of the sulcus Rolandi, or centralis, draw (according to Thane, Horsley, Dana) a connecting line corresponding to the middle of the skull or to the sagittal suture, between the root of the nose and the external occipital protuberance, divide this line in two, and about two centimetres behind this point is the upper terminus of Rolando's fissure. It is more exact to measure off a distance corresponding to 55.7 per cent. of the distance between the glabella and the inion. The fissure passes from here downward and frontward, and forms with the sagittal line an angle of about 67° (or, on an average, 60° to 70°). Wilson has recommended a particular instrument for these measurements.

FIG. 185.



Topographical relations between the upper cerebral surface and the cranium. *c*, central fissure; *HC*, lower, *VC*, posterior and anterior central convolutions; *S*, *Si*, and *Sii*, fossa Sylvii; p^1 and p^2, upper and lower parietal lobes; *O*, occipital lobe; *Cb*, cerebellum; *T*, temporal lobe; *F*, frontal brain. (After Ecker.)

In operations on the skull, the surgeon should keep about two centimetres away from the sagittal suture in order to avoid injuring the longitudinal sinus.

To determine the lower terminus of the fissure of Rolando, one should mark, according to Poirier, first the upper angle of the zygomatic process of the temporal bone, and should then erect, upon this as a base, a vertical line, which passes upward exactly in front of the tragus, between it and the posterior border of the maxillary articulation. Upon this line, seven centimetres above the external auditory meatus, lies the desired point.

The lower terminus in children is found by connecting the external auditory meatus with the sagittal suture by a vertical line and measuring off fifteen millimetres below its centre.

Bennet and Godlee, in accordance with the views of Reid and von Bergmann, used another method for the projection of the sulcus centralis upon the cranial surface. I have tried it myself, and, with others, have found it very useful. Upon the sagittal line (connecting line between the root of the nose and the external occipital protuberance) a vertical line is erected corresponding to the anterior border of the external auditory meatus, about two inches behind this a second vertical line parallel to it is drawn which touches the posterior border of the mastoid process. The point where this intersects the sagittal line corresponds to the upper end of the sulcus centralis. The lower is found upon the anterior vertical about two inches over the external auditory opening or about two inches above the upper border of the external auditory meatus. Numerous apparatus (cyrtometer, encephalometer, cranio-encephalometer) have been constructed upon the basis of these reports. They are, however, not of much use, and can be replaced by exact measurements with small strips of court-plaster, which are adapted to the cranium according to the rules laid down above.

If the position of the fissure of Rolando has been determined, it is not difficult to project the different areas of the motor zone, etc., upon the skull. The anterior central convolution begins below the anterior lower angle of the parietal bone. The division of the fossa Sylvii into the anterior and posterior arm lies, according to Merkel, about four or four and a half centimetres above the middle of the zygomatic arch. The temporal muscle covers the whole temporal lobe or its convexity, and the fossa Sylvii and the entire lower frontal convolution lie hidden under it also (Merkel).

Chipault has lately recommended a simple method for the projection of the chief fissures upon the upper cranial surface.

HISTOLOGIC CONSTRUCTION OF THE CEREBRAL CORTEX.

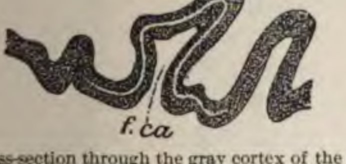
At many places on the brain cortex an examination with the naked eye suffices to recognize in a cross-section layers parallel to the upper surface. These are most distinct and constant in the neighborhood of the calcarine fissure, in which a white stria separates an external gray layer from an inner yellowish-gray one. (Fig. 186.) This bundle of Vicq d'Azyr corresponds to the less distinct Gennari's or Baillarger's stria at other places.

Microscopical examination of the cortex, which is not everywhere histologically the same, but which reveals a basic plan, has enabled us to recognize an arrangement of nerve-cells (and nerve-fibres) which renders it possible to differentiate a number of *layers*, which, it is true, are not sharply demarcated, but which are characterized by a particular type of cell predominant in each (Figs. 187–190).

Under the pia there lies a layer of neuroglia, which contains a few *small cells*. Upon its outer part nerve-fibres run parallel to the upper surface: the tangential fibres. In the deeper parts of this layer appear numerous *round cells*. The next layer is that of the *small pyramidal cells* (called by Audriezen the ambiguous layer).

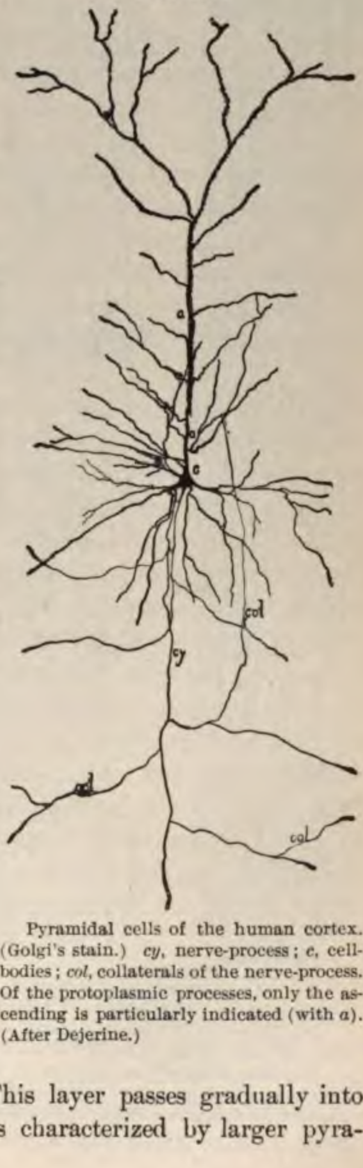
They are named from their pyramidal form, the apex of the pyramid is directed outward, from its base there arises an axis-cylinder process which dips into the gray matter (Fig. 187). This layer passes gradually into that of the *large pyramidal cells*. It is characterized by larger pyra-

FIG. 186.



Cross-section through the gray cortex of the cerebrum near the calcarine fissure. *f.ca*, calcarine fissure. The white line is the bundle of Vicq d'Azyr. (After Schwalbe.)

FIG. 187.



Pyramidal cells of the human cortex. (Golgi's stain.) *cy*, nerve-process; *c*, cell-bodies; *col*, collaterals of the nerve-process. Of the protoplasmic processes, only the ascending is particularly indicated (with *a*). (After Dejerine.)

FIG. 188.

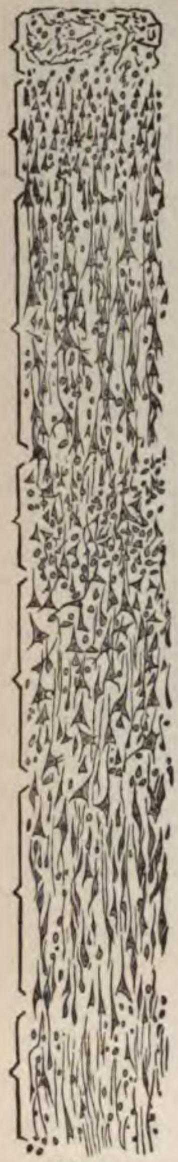


FIG. 189.

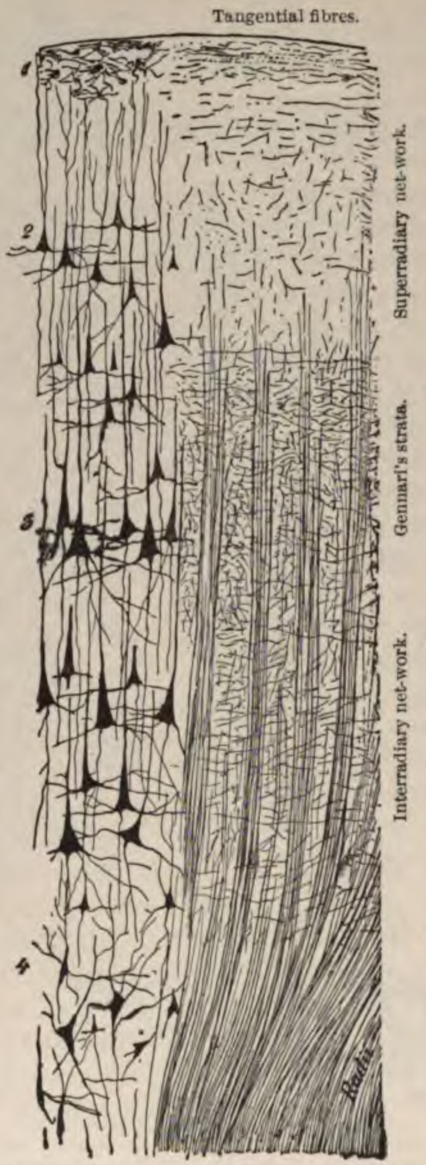


FIG. 190.

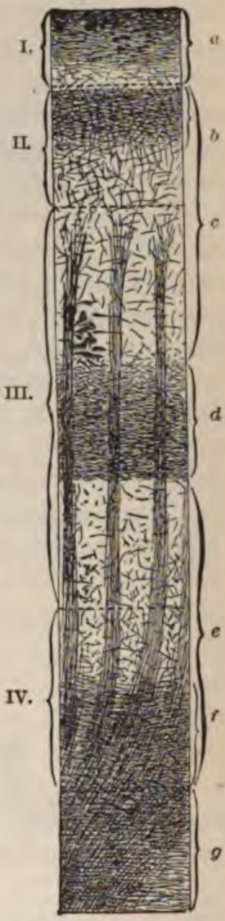


FIG. 188.—Schematic presentation of the layers of the cerebral cortex. Region of the first occipital gyrus. (After Bevan Lewis and Gowers.)

FIG. 189.—Section through a frontal convolution. To the right, stained with Weigert's stain; to the left, after Golgi's method. Fibres shown on the right; cells on the left. (After Edinger.)

FIG. 190.—Schematic presentation of the cortical fibres. *a*, tangential fibres; *b*, Bechterew's-Kaes's strata; *c*, superradiary net-work; *d*, Baillerger's strata; *e*, interradiary net-work; *f*, Meynert's intercortical association fibres; *g*, subcortical association fibres. (After Dejerine.)

midal cells arranged in rows, whose size increases as we pass inward. In addition, the medullated nerve-fibres now become more numerous, intersecting the strata in an almost vertical direction.

The deepest layers contain in the region of the central convolutions particularly of the *paracentral* and the *anterior central convolution*, very large ganglion cells,—Betz's *giant cells*. Following upon the layer of large pyramidal cells comes one containing smaller cells, at whose lowest border numerous *spindle cells* appear. This part has also been called the spindle-cell layer, or the layer of polymorphous cells. In addition, a number of small polygonal cells are scattered over all the layers of the cortex. We can therefore differentiate four, five, or more layers of the cerebral cortex.

Schlapp speaks of a five-layer and a seven-layer type, and is inclined to refer the arrangement to the functions of the cortex, an attempt which had already been made by other authors.

The dendritic processes of the ganglion cells comprise a large part of all layers, as also the axis-cylinder processes and their accessory branches. In addition, fibres arising in the medullated layer are found in all parts of the cortex.

Edinger distinguishes the following strata of fibres (Fig. 189): 1. Radii, radiary fibres or medullary radiations; 2. Interradiary net-work, consisting for the most part of parallel fibres on the upper surface; 3. Superradiary net-work; and 4. Tangential fibres. At the border, between the superradiary and interradiary net-work, the latter thickens to become Gennari's or Baillarger's stria, and, in the calcarine fissure, the bundle of Vicq d'Azyr. It has been assumed that the first is for the most part formed of processes from the lateral branches of the processes arising from the pyramidal cells. Bechterew has been able to separate another medullated stria in the inner cortical layer below the tangential fibres (Fig. 190). Fibres are also found in the deepest parts of the cortex, which can partly be followed from one gyre to another (association fibres, Fig. 190). The observations of Kaes and others have shown that the number of medullated fibres in the cortex increases considerably after birth, and even during the first half of life. In the first few months of life only a few areas of the cortex contain medullated fibres.

LOCALIZATION IN THE BRAIN CORTEX.

The individual parts of the cerebral surface are not of the same value, but are of different physiologic importance. Every *cortical area* which represents a *definite function* we call a *centre*. The significance, position, and limitations of the centres have been partly secured by experiments, principally by the discoveries of Fritsch and Hitzig, followed by Ferrier, Munk, Horsley, and others, but chiefly by clinical and pathologico-anatomic observations.

Motor Zone.—The motor zone comprises the region of the motor centres. These extend over both *central convolutions* and the *paracentral lobes.* (Compare Figs. 191 and 192, and 183.)

It is not known just how far the motor area extends into the neighboring parts of the frontal and parietal lobes. It seems that the posterior part of the frontal convolution in man belongs to this area. Some authors—for instance, Monakow—also include the anterior portion of the parietal lobe in the motor zone. This section, however, plays, in comparison with the central area, but a subordinate part.

Within the above-named area are found the centres for the musculature of the opposite side of the body, so divided that that for the *facial* and *hypoglossus* (probably also for the motor *fifth*) is situated in the lower third or fourth of both, but particularly of the anterior central convolution; while the *arm centre* takes in the middle third or the middle half of both, and in the anterior one extends somewhat higher than in the posterior. The upper part of the central convolutions, particularly the anterior, is taken up by the *leg centre*, which, however, extends also into the paracentral lobes.

Though these centres are by no means sharply defined, and shade into one another, recent observations, particularly those noted in modern cerebral surgery, have shown that within the individual centres a still more minute differentiation is possible, in that each muscular group or each muscle function corresponds to a special cortical area.

It is not improbable, therefore, that a close analogy exists between the localization of motor centres in the brains of man and the ape (Horsley), but it cannot be claimed that a detailed arrangement is as yet firmly established. Nevertheless, it seems to us necessary to include here for comparison the facts established for the brain of the ape (Fig. 191).

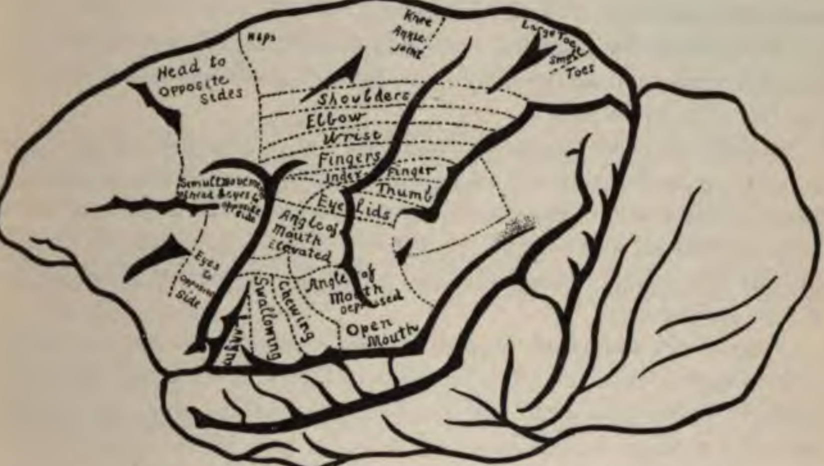
The observations upon which the figure is based show that we are not dealing with separate centres for single muscles, but for simple movements.

We can regard it as established that within the arm centre in man, also, the zone for the thumb and index finger lies the lowest (Fig. 192); that following upon this above is that for the other fingers and for the movements of the hand; while the field for the shoulder muscles is found at the highest pole.

The anterior central convolution, to all intents, has a greater part in motor innervation than the posterior. The centre for the extensors of the hand seems to lie in it, opposite to the posterior end of the frontal median gyre.

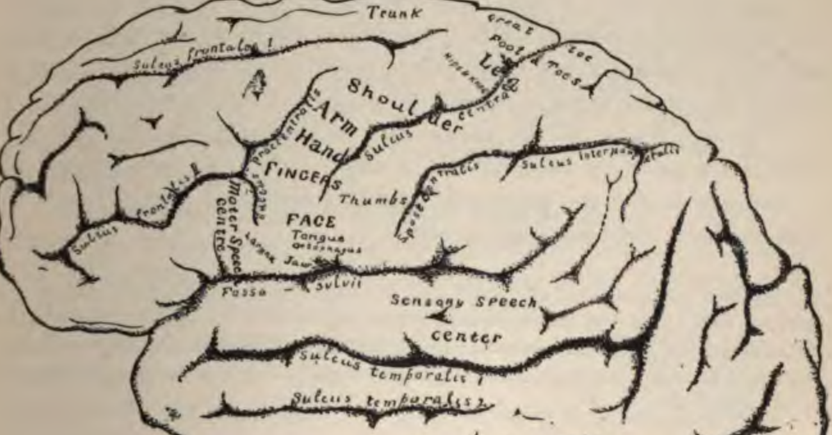
We have no exact knowledge of the arrangement of the leg centre.

Fig. 191.



Motor region of the cerebral cortex in apes. (After Beevor and Horsley.)

Fig. 192.



Motor region on the convexity of the cerebral cortex in man. The words in heavy type denote the established centres; the others are still *sub judice.*

It has not been settled whether the foot and toe or the upper thigh musculature lies in the lowest part. The views expressed have been very contradictory. My own observations indicate that the muscles supplied by the peroneus lie upon the convexity.

The centre for the extensor longus hallucis seems to lie in man also at the highest point of the central convolution.

The paracentral lobule has without doubt a considerable part in the cortical innervation of the lower limb.

In general, the finer and more complicated the movements of which the musculature innervated by these foci is capable, the more extensive the foci seem to be (Starr).

Numerous observations indicate that the centres for the laryngeal, masticatory, and deglutitory muscles localized by Krause, Semon-Horsley, Beevor, Réthi, and others in animals in the anterior lower end of the anterior central convolution, are situated in the same region in man, though this cannot be regarded as definitely established (Charcot and Pitres, and others).

The views expressed concerning the closer localization of motor centres in the lower end of the central convolutions or in the operculum, however, differ somewhat. Monakow's observations, that the posterior part of the third frontal convolution is the seat of the innervation of the tongue and partly of the larynx, is particularly questioned. Brissaud places the laryngeal centre where the horizontal ramus meets the ascending ramus of the fossa Sylvii.

The *facial centre,* it is certain, lies in the lower quarter of the anterior central convolution. The upper and lower facial are each assumed to have a separate small centre, though the question as to the innervation of the ocular facial is not yet definitely settled (see below).

The significance of the motor zone is cleared up by the fact that stimulation produces contractions in those muscles of the opposite side of the body which are governed from this area, and destruction of the same evokes a corresponding paralysis.

Stronger stimulation of the motor area, the farther down it is applied, causes contractions which pass from the muscular group involved first in regular order to others of the same side, and finally becomes generalized. Extended spasms may, therefore, be evoked from this cerebral area. Epileptogenous characteristics are only possessed by the motor zone.

It should, however, be noted that some of these centres govern not only the muscles of the opposite side, but also to a lesser degree similar ones of the homolateral side of the body (Broadbent); this is true of the muscles which are, as a rule, not used on one side only, but which act bilaterally symmetrically: the *masticatory, deglutitory, laryngeal, truncal,* and *orbicularis palpebrarum* muscles.

In accordance with this, unilateral stimulation of these centres is able to elicit bilateral contractions, as Krause, for instance, found to be the case with the internal thyreo-arytenoid, while a unilateral lesion does not produce a chronic paralysis.

This is more liable to result from a bilateral affection of these cortical centres, and then involve both sides. Monakow thinks the above-mentioned bilaterally acting muscles are, perhaps, chiefly innervated by subcortical centres and but slightly from the cortex.

The observations which indicate a cortical vocal cord paralysis (Dejerine, Rossbach, Eisenlohr) are very few and uncertain. The significance of the laryngeal centre has also lately again been questioned (Bechterew, Klemperer); while, on the other hand, Brissaud and Wallenberg have expressed the view that the corresponding cortical area for the most part or entirely governs the adductors of the *opposite* side. This question must, therefore, remain open, though I regard it as probable that the above-mentioned cortical centre for the larynx exists in men, and, as a rule, acts upon the muscles of both sides.

In reference to localization it cannot be denied that individual peculiarities may play a part, so that in some the motor centres of one hemisphere have more influence, a unilateral cerebral lesion being for this reason able to produce a paralysis of the entire tongue (glossoplegia), a deglutitory disturbance, etc. This is, however, very rare indeed.

As to the glossal muscles, v. Monakow believes that they, as far as they are used in speech, are innervated from one hemisphere, while the function of swallowing, eating, etc., is governed from both.

In the frontal lobes, especially the posterior part of the first and second frontal convolutions, we find centres for the movements of the eyes and the head (Ferrier, Horsley, Mott), particularly for moving the head and eyes to the opposite side. This appears to be true for man also, though in unilateral disease of this region corresponding paralytic symptoms do not occur.

Munk especially regards the frontal lobes as the seat of the innervation of the truncal muscles, Horsley and Schäfer the marginal convolution, while other authors (*e.g.*, Jackson) ascribe to the cerebellum a special influence upon the truncal musculature.

Centres for the innervation of the ocular muscles have also been found in other cortical areas,—for instance, in the lower parietal lobe (Wernicke) and the occipital lobe (Ferrier, Schäfer, Munk, Obregia). At any rate, a tract appears to arise in the visual sphere which conducts impulses for the fixing of the eyes; nevertheless, it is probable that the centre for *voluntary* fixation of the eyes belongs only to the areas of the frontal lobe which have been mentioned above.

The reference of the levator palpebræ superioris to the gyrus angularis (Grasset) is based upon very uncertain data. The same is true of Schäfer's view, that the destruction of the gyrus angularis in apes impairs the ability properly to calculate distances and evokes disorders of accommodation.

Vasomotor centres have also been located in the central convolutions and their vicinity, especially for the opposite side of the body. To these have been ascribed a regulating influence upon the heart and blood-vessels, upon heat production, and upon the vegetative nervous system (Eulenberg and Landois, Pitres, Franck, Bechterew), though these relationships, in man at least, are not yet clear.

The locations in man of the cortical centres for the bladder and rectum are not yet known. Experiments upon animals have led to the view that they are found in the neighborhood of the motor zone. In unilateral diseases of the cerebrum bladder disturbances are scarcely ever observed; in bilateral affections they would probably occur under certain circumstances, but satisfactory observations are lacking here also.

Several observations (Quincke, Kirchhoff, and others) tend to the view that the cortex contains *trophic* centres for the musculature, etc., of the opposite side, though this assumption is still hypothetical (see below).

The Speech-Centre.—The speech-centre is found in right-handed persons in the left hemisphere, and comprises the posterior part of the *third frontal convolution*, the *first temporal convolution*,—particularly its posterior two-thirds,—and perhaps the gyrus angularis (?). The posterior part of the third left frontal convolution contains the *motor* speech-centres, the region where concepts are converted into words (see the section upon aphasia). The posterior part of the right third frontal convolution seems to have a minor part in the act of speech, and in left-handed individuals is the chief centre. The first temporal convolution represents the *sensory* centre for speech,—*i.e.*, the place where the memory for the sound of words exists. According to Flechsig, this centre comprises also the horizontal convolutions of the temporal lobe which lies in the fossa Sylvii (the roots of the first temporal convolution). The part which the gyrus angularis is supposed to play in reading will be described later. A special writing centre probably does not exist—Charcot and Pitres located it in the foot of the second frontal convolution. It is doubtful whether the island of Reil is involved in the central acts of speech. It probably contains conducting paths, which connect the motor with the sensory centre of speech. Flechsig says that the island seems to be a centre which connects the motor and sensory cortical zones involved in speech into a unit.

We are less acquainted with the position of the *sensory centres* in the cortex than we are with the motor. Experimental observations, as also a number of clinical and anatomic observations, indicate that they are closely connected to the motor centres, and are partly or entirely identical.

The same view is furnished by the teaching that the real cause and origin of movements is due to *sensations of movement* (kinesthetic sensations).

Munk regards the motor region as the sensory zone. In diseases of this region sensory disturbances, as symptoms of irritation, have been observed. They need not, however, be present,—are, on the other hand, very slight in extensive paralysis of cortical origin,—and therefore it cannot be doubted that the sensory zone extends over a far larger area. The gyrus fornicatus is included in the sensory zone by Ferrier and Schäfer, with whom Horsley and Flechsig agree, while others, as Hitzig, doubt the existence of any relationship between this cortical region and sensibility.

At any rate, the sensory conducting tract (see below) passes for the most part into the central convolutions, particularly the posterior (Flechsig, Hösel), though its sphere of radiation probably extends to other areas, particularly the parietal lobes (v. Monakow). Atrophy of this tract, according to these authors, comes on only when the motor zone and the parietal lobes are diseased.

The opinion has been expressed that only sensations of touch and position are perceived in the central area, and that other sensory conceptions, particularly the sense of pain, should be referred to the gyrus fornicatus. V. Monakow refers stereognostic sensations particularly to the central area.

On the other hand, the *parietal lobe* has also been mentioned as a special centre for the transmission of the sense of position (Nothnägel, Luciani). Several clinical and anatomic observations (Vetter, Brasset, Monakow), and also an experimental observation of Starr, favor this view. Conclusive evidence is, however, still lacking. It is possible that the motor region as well as the parietal lobe is a factor in these sensory conceptions.

Finally, the significance of the motor region in regard to sensations is entirely doubted by Charcot and Pitres, while Brissaud has set up the hypothesis that the sensory zone of each hemisphere influences both sides of the body. This view, however, is not tenable.

Visual Centre.—The visual centre is located in the occipital lobe (Monk). It includes the calcarine fissure (according to Henschen exclusively) and the cuneus. Opinions differ concerning its further extent (v. Monakow, Flechsig, Vialet, and others). Probably the gyrus fusiformis (lateral occipito-temporal), lingualis (median occipito-temporal), and perhaps the first occipital convolution should be included.

Disturbance of the visual centre evokes bilateral hemianopsia of the opposite side. The rest of the cortical area of the occipital lobe—

probably the left angular gyre also—seems to be a factor in the valuation of the concept appearance of facial impressions, and in the widest sense of the word belongs also to the visual centre. The cortical layers of the convexity of the occipital lobe, according to Wilbrand, comprise a visual memory centre where the memory pictures of facial conceptions are combined (see Soul-blindness). It is questionable whether particular areas or layers should be separated for the senses of space, light, and color.

Diseases of the optic nerve which have existed many years may produce under certain circumstances an atrophy of the occipital lobe. Von Leonowa found in anophthalmia disappearance of certain cell-groups in the calcarine fissure.

The relationship between the visual centre and the optic conducting tracts will be discussed later on.

The view that, in addition to the occipital a further visual centre exists in the gyrus angularis, connected with the entire retina of the opposite eye, so that disturbance of it causes *blindness* in the eye of the opposite side, is no longer tenable, even though Seymour Sharkey has lately argued in favor of it.

A few experimenters believe that the macula of each side is represented in both visual centres (Wilbrand, Gowers, Knies), as unilateral diseases of the occipital lobe do not generally impair the function of the macula. It also happens that the seat of direct vision is spared in bilateral disease (Foerster, Sachs, and others). Other factors must therefore be present,—for instance, a better vascular supply to this region, as Foerster assumes. V. Monakow believes that the ganglion cells for the macula are particularly numerous in the subcortical centres, and raises the question whether the macula is not perhaps represented in the whole visual area.

Nothing definite is known concerning the seat of the **centre for smell.** A few observations indicate that the *gyrus uncinatus* governs such a centre. Diseases of this area may evoke anosmia of the same or of both sides.

A centre in the anterior part of the gyrus fornicatus has been supposed to be for the **sense of taste,** but this is not yet settled.

Paget has lately located centres for the sensations of hunger and thirst on the basal surface of the temporal lobe.

The auditory centre lies in the cortex of the temporal lobe in the upper convolution. Flechsig believes that that part of the first temporal convolution which is hidden in the fossa Sylvii, the horizontal convolution, forms the chief auditory centre.

Pathology teaches that the impairment of hearing which occasionally occurs in the ear of the opposite side in diseases of the temporal lobe is of temporary duration. It can be assumed that the centre of each hemisphere is connected with both acoustic nerves, so that impairment of one is soon compensated for by that of the other side.

The functions of the other cortical regions are not exactly known.

The act of thinking is probably connected with the *entire cortex*. The *frontal lobes*, however, seem to take precedence in the higher psychic functions.

Flechsig's division of the cortical region into centres of sense and of association is not accepted by the majority of authorities. He includes under the latter sections of the parietal, temporal, and frontal lobes, which are characterized by not receiving any tangential fibres, or conducting tracts from the periphery (organs of sense, spinal cord), but are only connected to the other cortical areas by associative fibres. Sachs particularly has combated this teaching, and has expressed the opinion that projection and association fibres arise from every cortical area.

CONDUCTING TRACTS.

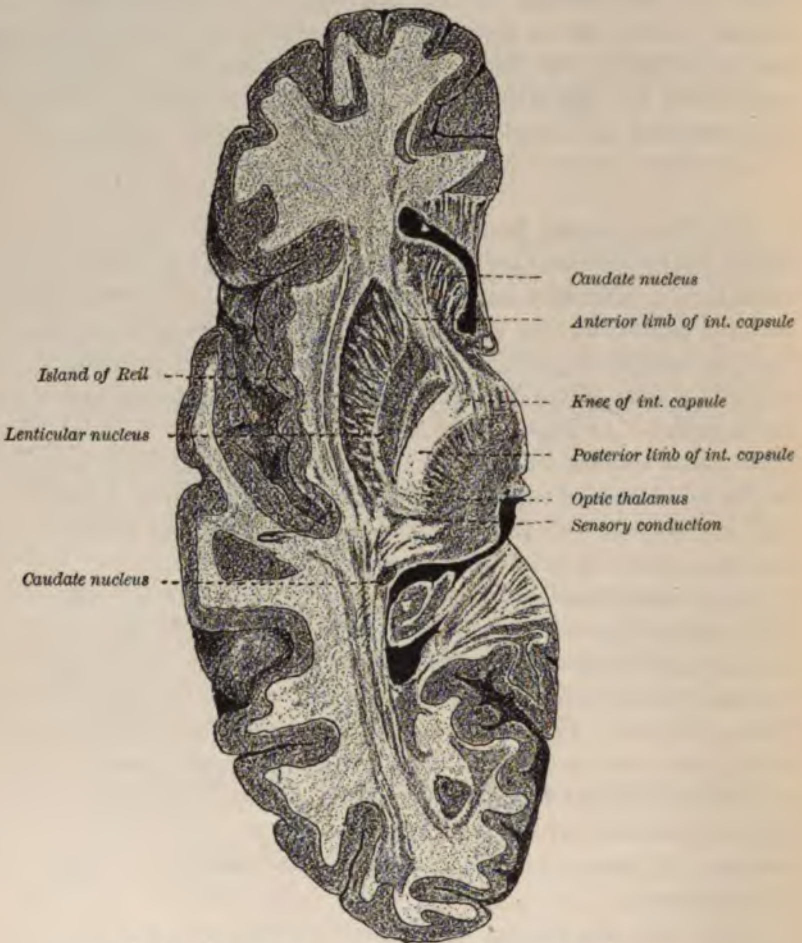
The fibres coming from the motor centres converging from all sides tend to pass to the inner brain and form a part of the coronary or tegmental radiations. After they have taken up a still relatively large area in the centrum semiovale, they force their way into the inner part of the brain, into the *internal capsule*, where they occupy a very narrow space. The medullary tract of the *internal capsule* consists of an anterior and a posterior limb. As Figs. 193 to 196 show, the first lies between the *caudate nucleus* and the *lentiform or lenticular nucleus*, the latter between the *thalamus opticus* and the *lenticular nucleus*. The *pyramidal tract* extends into the anterior two-thirds of the posterior limb, and it appears that the fibres from the facial centre are mostly in front, near the knee or in it, while back of this are found those for the arm, and still farther back those for the leg, without, however, the separation being a sharp one. Certain bundles of fibres for the tongue, masticatory and laryngeal muscles have also been differentiated in the knee-part of the capsule (Horsley, Semon, Beevor). The tracts are apparently arranged behind one another in the same manner as are their centres in the motor zone; though the separation is by no means very complete, so that lesions in the internal capsule generally involve all the motor fibres. It is also questionable whether the facts concerning localization established in animals hold true for man.

The path for speech, according to Monakow, positively passes through the knee of the inner capsule. The anterior limb contains among others the so-called frontal pontile tract (see below).

The pyramidal tract passes from the anterior capsule to the foot of the cerebral peduncle, and assumes here (see scheme, Fig. 197) the space included between 2 and 3. The conducting tracts for the motor cranial nerves lie probably towards the inner side from those for the extremities, while fibres are said to run in the median and lateral area which arise from different regions of the cortex, and practically end in the gray pontile nuclei.

Fibres are found in the median fasciculus of the foot of the pedunculus cerebri which arise in the frontal brain (Flechsig, Monakow)—according to Dejerine, from the third frontal convolution and the foot of the central convolutions—and pass through the anterior limb of the internal capsule to reach the pons. Some believe that a part of these fibres end in the thalamus opticus. Nothing definite is known concerning the significance of this tract; it has been claimed that it serves to carry psychic, affective impulses to deeper centres (Brissaud's psychic bundle). On the other hand, the opinion has been

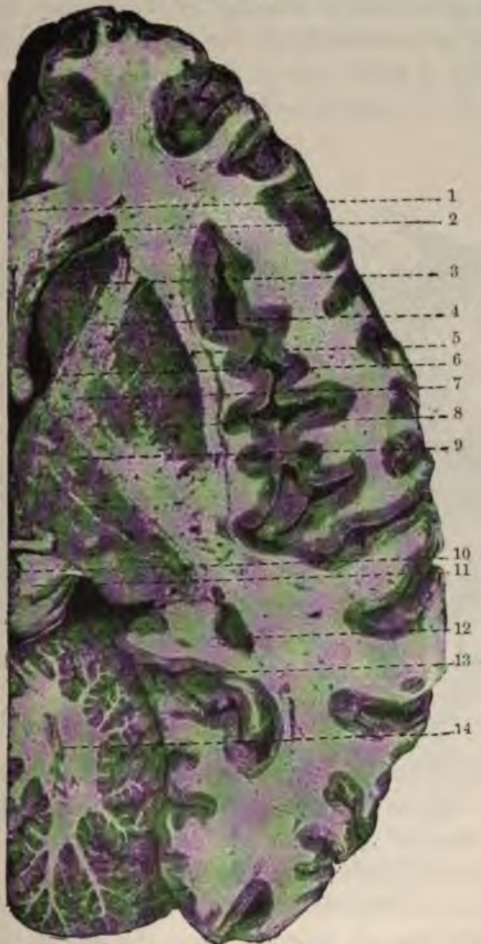
FIG. 193.



Horizontal section through the human brain. (Brissaud.)

expressed that they connect the cerebrum with the cerebellum by the intercalation of gray masses, which lie in the pons (see below). According to Dejerine it contains fibres which arise in the speech-centres and cortical centres of the motor cranial nerves, while others place these tracts somewhat more externally (at 2) or describe a special tract for this function, the so-called lower lemniscus (Fussschleife), which passes from the foot of the cerebral peduncle gradually into the median part of the lemniscus (Spitzka). This would make it a cortico-nuclear tract of the motor cranial nerves. The lateral bundle of the pes pedunculi is said to arise from the temporal and occipital lobes, according to Dejerine and Monakow only from the first. The pes pedunculi cere-

FIG. 194.

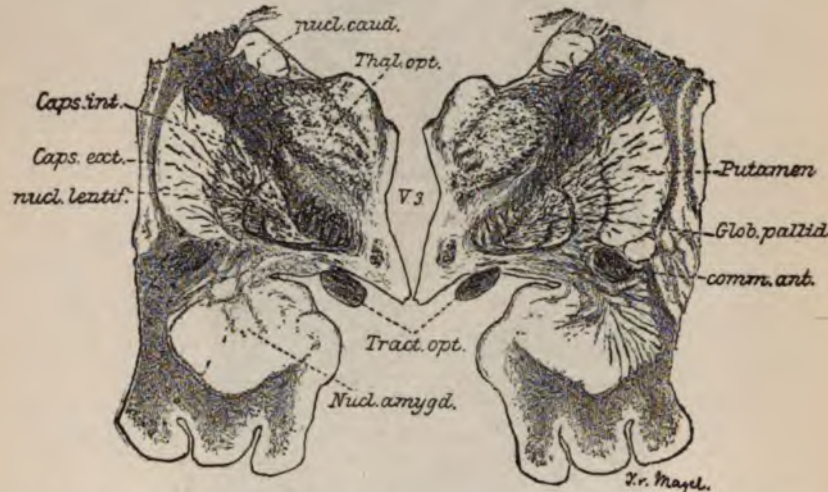


Horizontal section of a human brain. (From a photograph of a recent preparation.) 1, corpus collosum; 2, anterior horn of the lateral ventricle; 3, caudate nucleus; 4, internal capsule (anterior limb); 5, lenticular nucleus; 6, columna fornicis; 7, internal capsule (knee); 8, internal capsule (posterior limb); 9, optic thalamus; 10, pineal gland; 11, corpus quadrigeminum; 12, inferior horn of the lateral ventricle; 13, parieto-occipital fissure; 14, nucleus dentatus of the cerebellum. (After E. Flatau.)

bri is, therefore, formed from neurons or nerve-processes which arise in the cerebral cortex.

Figs. 203, 206–215 show the further course of the pyramidal tract. In the pons it is covered by a superficial layer of transverse fibres and is divided into a number of bundles by the pontile fibres which pass through it. In its course through the pons and oblongata it gives off fibres at all heights, which pass to the raphe, decussate there, and after decussation reach the appropriate *nucleus of a motor cranial nerve.* It has not been as yet possible, however, to trace all these fibres throughout their course. As far as we know, the decussation takes place not far above the nucleus. Most of the motor conducting tract, that for the extremities, the cortico-spinal tract, passes to the lower part of the medulla oblongata, where an *incomplete decussation* occurs (Fig. 198). The motor conducting tracts descend, therefore, from the centres to the spinal cord, without being interrupted by gray matter. Gattel and Monakow have given most accurate descriptions of their position and order in the pons.

FIG. 195.



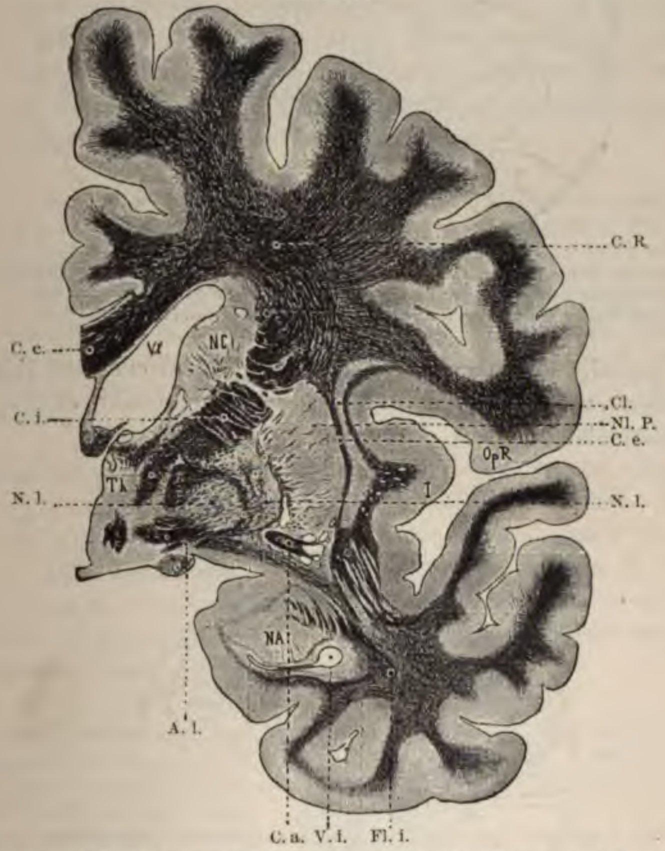
Part of a frontal section through the brain-stem near the internal capsule. (Pal's stain.)

The facial tract, which, in the internal capsule and probably also at the foot of the cerebral peduncle, lies directly against the pyramidal tract, leaves it in the anterior part of the pons to reach the nucleus of the other side after decussation in the raphe. Some of the fibres may pass to the nucleus of the same side (Hoche).

The fibres coming from the *hypoglossal* centre are supposed to pass over the lenticular nucleus, and to lie in the internal capsule between the facial tract and that for the extremities. They separate within the pons or the medulla oblongata from the pyramidal tract, and pass on the median side of the lemniscus backward and upward to the raphe to sink into the nucleus after decussation. Here, also, some of the fibres appear to pass to the nucleus on the same side.

We know little that is reliable concerning the motor path for speech. It probably also passes through the internal capsule (knee) and the foot of the cerebral peduncle, where it is said to lie externally to the hypoglossal tract (?) (Raymond and Arthaud). Other fasciculi—the lower lemniscus, the accessory lemniscus of Bechterew, or the lateral pontine fasciculus of Schlesinger—have been thought to be speech tracts (Mingazzini).

Fig. 196.

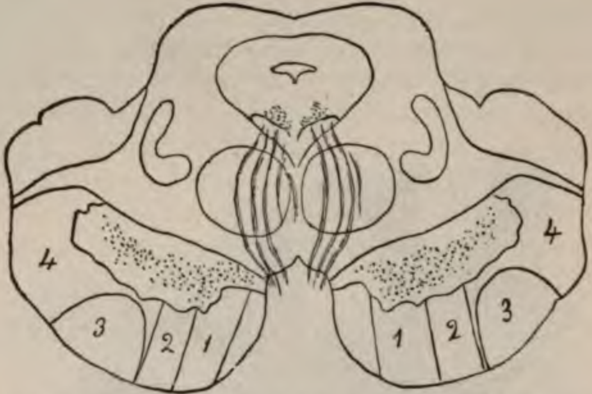


Frontal section through a human brain. Weigert's stain. (After Dejerine.) OpR, operculum; NC, caudate nucleus; Vl, lateral ventricle; I, insula (island of Reil); NA, nucleus amygdalæ; C.R., cornea radiata; Cl., claustrum; Nl.P., lenticular nucleus (putamen); C.e., external capsule; N.l., lenticular nucleus; Fl.i., inferior longitudinal fasciculus; V.i., inferior ventricle; C.a., anterior commissure; A.l., ansa lentiformis; C.i., internal capsule; C.c., corpus callosum.

Our knowledge of the course of the *sensory conducting tracts* is especially unreliable. Recently, however, some light has been shed upon the question.

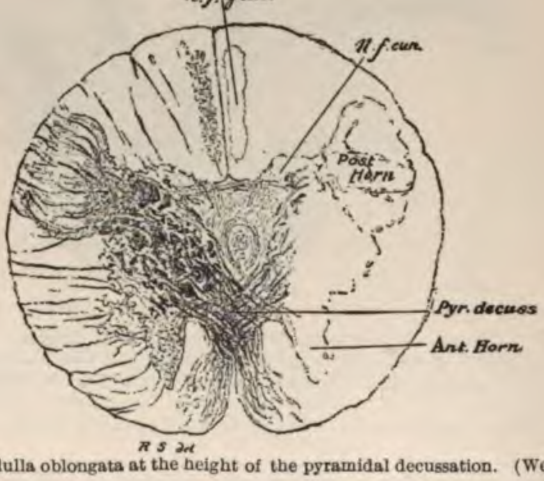
If we seek to follow these tracts from the spinal cord to the brain, it must be remembered (page 103 and following) that part of these tracts, those passing up the posterior columns, end in the terminal nuclei of the medulla oblongata, the nuclei gracilis and cuneatus (Fig. 198), while others, which decussate in the spinal cord, probably ascend by way of the anterolateral tracts.

FIG. 197.



Tracts of the foot of the cerebral peduncle (schematic). 1, fibres from frontal brain to the pons; 2, motor conducting tracts of the cervical nerves; 3, motor conducting tracts for the extremities; 2 and 3, pyramidal tracts; 4, fibres from the temporal and occipital brain to the pons.

FIG. 198.



Section of the medulla oblongata at the height of the pyramidal decussation. (Weigert's stain.)

Fibres arise from the posterior column or terminal nuclei, and pass to the raphe as internal arciform fibres, and decussate there (Fig. 199). This upper decussation, which is also called the *sensory* or the *lemniscus* decussation, contains without doubt most of the sensory conducting tract, which next passes to the space between the olive and the raphe,—the midolivary strata,—and, as the median or chief lemniscus, reaches the brain. (Compare Fig. 200 as well as 215 back to 206.) It forms, therefore, a sensory conducting tract of the second order.

The tract which ascends in the anterolateral column, and which decussates in the spinal cord, joins the sensory tract. Some consider it as mixing with the lemniscus fibres, and taking a position dorsad to it, in the space between the olivæ, while others regard it as passing to the reticular field, or formatio reticularis. It is probable that this tract serves especially for the conduction of the pain and temperature senses, while the fasciculus running in the lemniscus decussation serves to transmit sensations of position and tactile sensations.

Observations of Wernicke, Senator, Goldscheider, and Bogatschow have made it probable that the tracts transmitting the muscular sense are separated in the oblongata from the others, and are found in the part of the midolivary layer lying next to the raphe. As this bundle only decussates after reaching the oblongata, a unilateral lesion of it, according to the height of the seat of the disturbance, can elicit a disturbance of the sense of position (and dynamic ataxia ?) upon the opposite or the same side.

Van Oordt places the tract conducting taste impulses in the ventral median part of the formatio reticularis. We have, however, not yet passed beyond the field of hypotheses in these questions.

There are two other sensory tracts ascending from the spinal cord: the *direct cerebellar* tract and the *ascending anterolateral* tract. The first enters the corpus restiforme without decussation and passes to the cerebellum. This tract probably transmits sensory impulses to the cerebellum, from the spinal cord or periphery, which regulate co-ordination. Lesions of it appear to produce incoördination (cerebellar ataxia and homolateral dynamic ataxia ?).

Gowers's tract separates from the cerebellar tract in the medulla oblongata, and probably passes to the region of the origin of the fifth nerve in the pons (Hoche, Tooth), and then enters the cerebellum (Auerbach, Mott). Some authors have it terminate in the lateral lemniscus.

Fibres from the nuclei of the posterior column also reach the corpus restiforme and cerebellum by means of the posterior external arcuate fibres (Hösel).

Bechterew, according to whose observations a larger part of the fibres which run centralward in the anterolateral residual columns are continued in the formatio reticularis, has them terminate for the most part in the gray nuclei of it (inferior and superior central nucleus; tegmental reticular nuclei, etc.), and from here extend to the optic thalamus.

There is also a set of fibres which pass from the cerebellum through the restiform body into the oliva of the opposite side (see below). We know nothing definite concerning the significance of this cerebello-olivary tract.

The same is true of a set of fibres in the formatio reticularis described by Bechterew, the central tegmental fasciculus (*centrale Haubenbahn*), which lies dorsad to the olivæ, and ascends from them to the midbrain (Figs. 209 and 210). We will refer later on to these tracts, which connect the cerebellum with the oblongata, cerebrum, and spinal cord.

Observations as to the further course of the chief lemniscus (median, upper lemniscus) do not agree. It is known that the sensory tract passes through the posterior limit of the internal capsule, and lies here in the retrolenticular part, behind the motor path (*carrefour sensitif*).

It is also settled that these sensory tracts pass wholly or for the most part into the central convolutions, especially the posterior, the paracentral lobe, and the parietal lobe.

Opinions differ, however, as to the direction; some (Flechsig, Hösel) believe that most of the lemniscus tract passes directly to the cortex, while a majority of observers (Monakow, Mahaim, Dejerine, Bechterew, Mingazzini, Schlesinger) think differently. They are inclined to believe that it enters different nuclei in the thalamus, especially the ventral, and from here passes to the cortex by new neurons. According to this, the tract consists of at least three neurons: (1) the spino-bulbar; (2) the bulbo-thalamic; and (3) the thalamo-cortical. There is a possibility that dendraxones are intercalated between these, as Monakow assumes.

Many facts seem to indicate that some of the sensory tracts are interrupted in the lenticular nucleus and the deeper-lying masses before they ascend to the cortex, though a relationship between the lenticular nucleus or its loop to the lemniscus is denied by some. Bechterew allows a part of the lemniscus to pass into the subthalamic body, globulus pallidus, reticular nucleus of the tegmentum, and the optic thalamus. Only the so-called accessory lemniscus, which he regards as the supranuclear path of the sensory cranial nerves, and the lower lemniscus enter the cortex directly, according to his ideas. The lemniscus probably contains short tracts also, which interconnect different heights of the pontile-oblongata region.

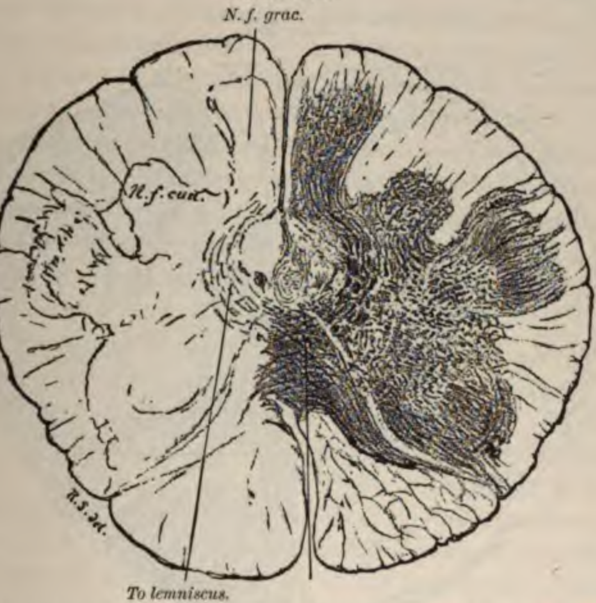
A set of fibres cutting directly through the internal capsule, called the tegmental radiation, which ends in the posterior central convolution and in the parietal brain, and passes from there to the central ganglia and to the subthalamic region, is included by some investigators in the sensory conducting tract, though this is still *sub judice*. V. Monakow particularly has combated this view, and has named other fasciculi tegmental radiations, which have nothing to do with the lemniscus.

We have, then, the following main path for sensory conduction: (1) Posterior column—Nuclei of the posterior column—Lemniscus or fillet decussation and the internal arciform fibres—Midolivary strata—Chief lemniscus, and from here directly to the cortex, or after interruption in the thalamus, or perhaps also in the lenticular nucleus. (2) A second tract, which ascends in the anterolateral column of the spinal cord, and joins the chief lemniscus in the medulla oblongata, or passes by means of the reticular field to the midbrain. It also ends in the cortex (central convolutions, parietal brain, or gyrus fornicatus).

In the sensory conducting tract there appears to be still another separation into different fasciculi for the various sensory qualities at the place where it passes through the posterior limb of the internal capsule. Kirchhoff lately attempted to limit the conducting tract for the sensation of pain. I observed in a case of shot-wound of the brain, in which everything indicated a lesion of the carrefour sensitif, an isolated thermanesthesia in a part of the opposite half of the body (see below).

(3) A secondary conducting tract also comes from the sensory cranial nerves and their nuclei, decussates in the raphe, and passes to the cortex (Edinger, Schlesinger, Bechterew, Wallenberg). We know little that is definite concerning the course of these nucleocortical paths of the sensory cranial nerves. Perhaps they pass into the chief lemniscus or traverse in part the reticular field.

FIG. 199.



Cross-section through the medulla oblongata at the height of the sensory decussation. (Weigert's stain.)

It appears certain from the above that sensory stimuli may follow one of many paths, and it is therefore probable that after impairment of a chief tract they may be transmitted by secondary paths.

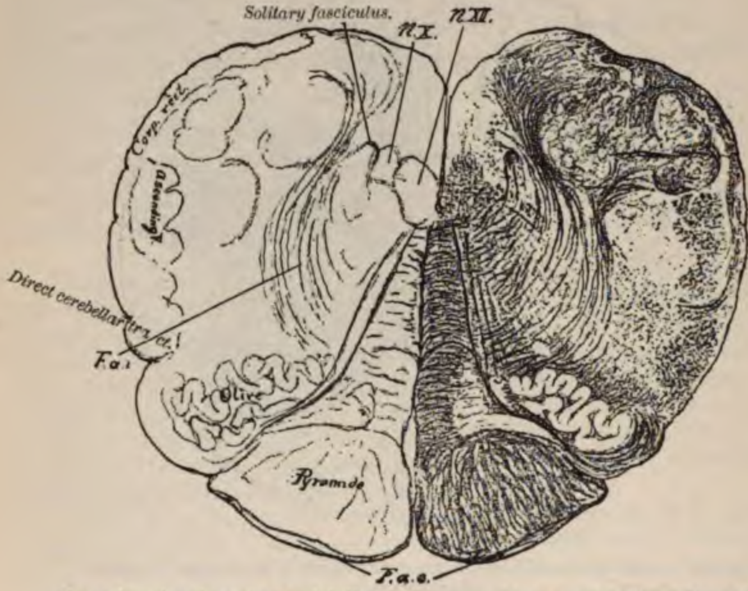
The sensory paths are probably also in connection with the motor nuclei of the cranial nerves, probably also with the central ganglia, and this is perhaps the way in which reflex actions may occur.

The anatomy and physiology of the *cerebellum* will be discussed in a later chapter. We are here interested only in the tracts which connect it with the cerebrum and spinal cord. They are found in the cerebellar peduncles.

The lower one, the *restiform body*, contains the cerebellar tracts, also fibres which, coming from the posterior columns, enter the cerebellum, and also the cerebello-olivary tract, which joins the cerebellum to the olivary body of the opposite side, and a tract which passes from the nerve-nuclei of the medulla oblongata (vestibular nerve, dorsal acoustic and Deiters's nucleus) to the cerebellum.

The *upper cerebellar peduncle*, brachium conjunctivum, the tegmentary cerebellar tract, arises in the cerebellum, principally in the corpus dentatum, decussates completely or partially under the gemina (corpora quadrigemina), and then enters the red nucleus. It is said to be connected by fibres with the optic thalamus and the cortex of the central parietal area.

FIG. 200.



Frontal section through the medulla oblongata. *F.a.i.*, internal arciform fibres; *F.a.e.*, external arciform fibres. (Pal's stain.)

The *middle cerebellar peduncle* consists chiefly of fibres which pass from the cerebellum to the gray pontile nuclei of the opposite side (Ramon y Cajal, Bechterew, Mingazzini).

Other fibres coming from the cerebrum (frontal lobe) descend in the median part of the foot of the cerebral crus, the terminal fibrils of which surround the cells of the gray pontile nuclei.

It has been assumed that this frontal-cerebral-pontile tract forms a coherent system with the middle cerebellar limb of the opposite side, which joins the cerebrum (frontal brain) with the cerebellar hemisphere of *the opposite side.*

Our knowledge of all these tracts is, however, still very incomplete. The following conceptions seem to be established. In the lower cerebellar peduncle are found fibres which pass to the cerebellum, and which influence co-ordination and regulate impulses—unconscious sensations—sensations passing from the periphery into the cerebellum. We ascribe this to the cerebellar tract which transmits to the cerebellum sensations of position coming from the truncal musculature. Such impulses also come from the labyrinth, by means of the vestibular nerve. The purpose of the crossed cerebellar-olivary tract is not yet clear. Kölliker believes that it arises in the cerebellum and transmits impulses to the periphery which have a part in co-ordinated muscular action.

Bechterew includes the olives among the centres of co-ordination, and has impulses reach the cerebellum through the cerebello-olivary tract. The olives, according to his conception, are connected with the central tegmental tract of the same side, by means of which stimulations are transmitted to it, which pass downward from gray masses in the region of the third ventricle.

According to others, the cerebello-olivary tract forms a single system of fibres with the brachium conjunctivum.

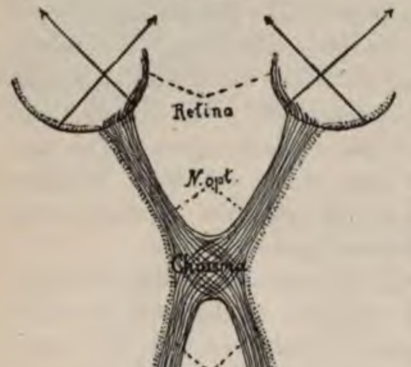
It has not been determined whether the brachium conjunctivum leads from or transmits to the cerebellum, or contains fibres for both purposes. It has been thought that the cerebellum takes part in the functions of the motor cerebral centres by means of this path, and in this way exercises a co-ordinating influence. On the other hand, it is possible that impulses may pass by this path from the motor zone to the cerebellum.

Kölliker, Bechterew, and others believe that tracts pass from the cerebellum through the middle peduncle, and, after passing through the gray nuclei of the pons and medulla, descend to the spinal cord, and as they reach the spinal muscle nuclei directly influence muscular co-ordination. This tract is said to be found in the reticular field and in the anterolateral column of the spinal cord. The pyramidal tract is also said to contain fibres of this category. This is, however, entirely hypothetical; a descending degeneration in the spinal cord, due to a lesion of the cerebellum, as was said to have been observed, for example, by Marchi, does not appear to occur in man.

We are also still in the dark concerning the significance of the middle foot of the cerebellar peduncle. It has been thought—L. Bruns has developed this hypothesis the most clearly—that the frontal brain, the highest volitional centre for the truncal musculature, transmits through it to the cerebellum impulses which exercise a certain degree of volitional control upon its co-ordinating impulses.

The connection of the cerebellum to the cerebrum seems to be entirely a crossed one (Russel, Luciani, and others).

FIG. 201.



Schematic representation of the visual conducting tract (without any regard having been paid to the relations in size between the different parts, etc.).

The *lateral fillet* or *lemniscus*, which arises at the height of the upper olivary body (Fig. 213), is probably a sensory conducting path of the second order which receives the acoustic impressions, especially from the acoustic nucleus of the opposite side of the medulla oblongata, and probably transmits them through the postgeminum (posterior quadrigeminum) and the internal geniculate body to the parietal lobe (Monakow, Bechterew, Held, Baginsky, and others). The cochlear nerve is regarded as the true auditory nerve. It ends in the ventral acoustic nucleus of the oblongata and in the acoustic tubercle.

The trapezoid fibres arise in these nuclei, especially the first, and pass to the upper olive of the same and, particularly, of the opposite side. Another neuron of the auditory tract arises here, the lateral (upper) lemniscus, which (in part) terminates in the postgeminum and the postgeniculum (internal geniculate body). Another tract then passes from here through the first temporal convolution to the cortex. The auditory tract therefore consists of at least four neurons.

Von Monakow, however, does not consider it definitely settled, that the internal geniculate bodies are auditory centres, and calls attention to the fact that it is this part of the brain which degenerates after disease of the temporal lobe.

THE VISUAL CONDUCTING PATH.

The optic nerve undergoes partial decussation in the chiasm. This is true, notwithstanding Kölliker, who has recently claimed to have found total decussation in man as well as in animals. The larger median fasciculus passes to the opposite side; the lateral remains on the same side. (Fig. 201.) The optic tract of one side is therefore in connection with the outer half of the retina of the same side, and the inner half of the retina of the opposite side.

The fibres of the optic tract terminate in the *pulvinar* of the optic thalamus, the *lateral geniculate body*, and the *anterior corpus quadrigeminum* (pregeminum).

The lateral geniculate body is the chief terminus of the optic fibres. Whether the optic tract enters the pulvinar and pregeminum directly, or whether the fibres entering here serve, as Henschen and v. Monakow assumed, only to transmit reflex movements (especially the light-reflex), is not known.

Von Bechterew claims to have proven experimentally, that lesions of the pregeminum produce blindness; observations on human beings have shown, however, that disease of this region does not necessarily produce any gross visual disorder.

This terminus, the *primary visual centre*, is connected with the *cortex* of the *occipital lobe* by *Gratiolet's optic radiations*, which pass through the extreme posterior part of the internal capsule, and from there into the occipital lobe. Most of this tract can be followed to the cortex of the cuneus. The optic radiations pass by the lateral side of the posterior horn; according to Henschen they pass the temporal lobes at the height of the second convolution and go through the deep white matter of the lower parietal lobule.

The fibres of the optic nerve arise for the most part from ganglion cells of the retina to end in the end-brushes of the primary optic centres. A second neuron arises in ganglion cells of these centres to pass to the cortex of the occipital lobe. The optic radiation, however, also contains fibres which arise in the cortex of the occipital lobe and pass to the primary optic centres. Nerve-processes are said to pass from these to the retina.

V. Monakow assumes that dendraxones (Schaltzellen) are intercalated between the first and second neurons of the optic tract.

Fibres are also said to pass from the occipital lobe to the ganglion cells of the nuclei of the ocular nerves.

In the occipital lobe, in addition to the optic tract, a number of other tracts have been described (Sachs, Forel, Dejerine, Vialet, and others).

These are supposed to be association tracts. Neither their anatomic nor physiologic existence has been absolutely determined (Flechsig).

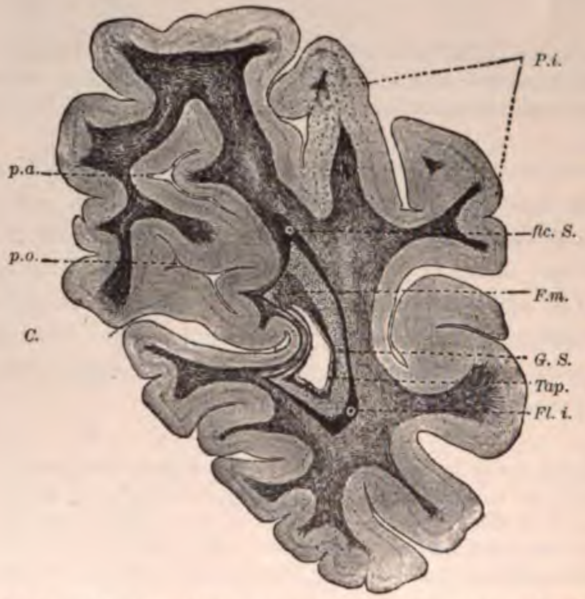
THE CENTRAL GANGLIA.

It was formerly thought that the *caudate nucleus* influenced movements of walking and running, but this has not been sufficiently proved.

The *lenticular nucleus*, especially the globulus pallidus, seems to be a passage-way for part of the sensory conducting paths; its significance is, however, not yet clearly known.

The *optic thalamus* is extensively connected with the cerebral cortex and with the conducting tracts which ascend from the spinal cord. Monakow, particularly, showed the relationship existing between certain nuclei of the optic thalamus and different cortical areas. The thalamus receives fibres from the great sensory tract; sensory impulses from the periphery to the brain pass through it. The optic tract is intimately connected with these ganglia (pulvinar). The sensory conducting path, the lemniscus, acts upon the optic thalamus, or rather ganglion cells in it, and these transmit the impulses to the cortex. Von Monakow considers all the nerves of special sense as being connected with the thalamus before they reach the cortex, and that lesions of the optic thalamus do not show it because they are connected with both sides of the thalamus. His views have not yet been confirmed.

FIG. 202.



Frontal section through the posterior part of the precuneus, through the calcarine and parieto-occipital fissures (Weigert's stain). *P.i.*, inferior parietal lobe; *flc. S.*, fasciculus transversus cunei (Sachs); *F.m.*, forceps major; *G. S.*, Gratiolet's visual radiations; *Fl. i.*, inferior longitudinal fasciculus; *C.*, calcarine fissure; *p.o.*, parieto-occipital fissure; *Tap.*, tapetum. (After Dejerine.)

Bechterew regards the optic thalamus and the quadrigemina as reflex-organs, as sensory conducting tracts not only lead to them, but tracts also arise in them which take a centrifugal course and act upon the muscular apparatus.

The functions of this part of the brain are as yet little known, notwithstanding all of these anatomic researches. It is not even positive that lesions of the pulvinar will produce hemianopsia; perhaps this only occurs in simultaneous injury of the external geniculate body. It is also not settled, whether diseases confined to the optic thalamus will produce anesthesia of the opposite side of the body, though, according to modern conceptions of the course of the sensory conducting tract, it should be expected.

Many investigators (Bechterew, Nothnagel, Brissaud) believe that the optic thalamus is a centre for *involuntary, automatic* movements, for the psycho-reflexes; that is, for movements which are not directly under the influence of the will. Lesions of the thalamus may, therefore, according as to whether they are irritative or destructive, increase the automatic, mimic movements, or cause them to disappear. Nothnagel showed, for example, that disease of the optic thalamus may be recognized by a crossed facial paralysis observed only in laughing, while the nerve still obeys the will. Involuntary movements have been often noticed in diseases of the optic thalamus. (See section on hemiathetosis, etc.) Are they due to a stimulation of a centre for involuntary movement? or does the stimulation of the sensory fibres excite the motor areas which express themselves by involuntary movements? Are there inhibitory fibres passing from the thalamus to the cortex, governing the motor region, interruption of which excites motor symptoms of irritation? or do the impulses not come from the optic thalamus at all, but from a lesion of a neighboring tract? All these questions cannot be definitely answered, though, with Monakow, I regard it as probable, that the motor symptoms of irritation are produced through excitation of centripetal fibres in the motor cortical zone.

Bechterew and his students found in animals centres in the thalamus for cardiac, gastric, and intestinal movements, and also for sexual actions. Vasomotor functions have been ascribed to it (Schiff, Lusanna, Sinkler); also secretory and trophic functions.

Anton assumes a sort of antagonism between the optic thalamus and the lenticular nucleus, in that disturbance of the latter produces an overbalance and thereby causes an increase of involuntary motion (?).

NUCLEI OF THE CRANIAL NERVES.

The nuclei of the cranial nerves, which are at the same time *trophic centres* for the motor nerves, lie in the floor of the third ventricle (posterior part), of the aquæductus Sylvii, and the gray matter enveloping the fossa rhomboidalis, and in part, also, the deeper part of the pons and medulla.

The nucleus of the oculomotor nerve arises in two nuclear chains in the pregeminum, under the aqueduct of Sylvius. It consists of a number of cell-groups.

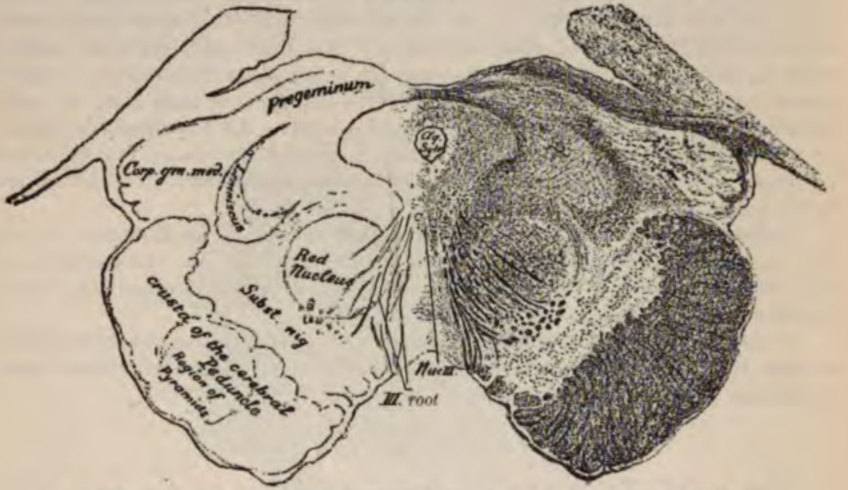
Attempts have been made by different authors to separate the cell-groups of the third nerve from one another.

Perlia speaks of a chief group which he has subdivided into four other groups, an anterior and posterior, dorsal and ventral. To these we must add a single central nucleus (Spitzka's sagittal nucleus), and Westphal-Edinger's groups in the anterior part of the nucleus. Ventrad, we find the anterior nucleus of Darkschewitsch just where the aqueduct of Sylvius opens into the third ventricle. Kölliker describes a chief nucleus, at the cerebral end of which a round nucleus branches off. The chief nucleus is subdivided into two parts, a dorso-lateral group with large cells, and a dorso-mesal group with small ones; and, in addition, a single central nucleus.

The schematic drawing (Fig. 204), after Siemerling, shows as the chief deep origin of the oculomotor nerve the lateral large-cell nucleus, but the central large-cell nucleus is also considered in the area of the nerve. The anterior nucleus of Darkschewitsch has probably nothing to do with the oculomotor (Cassirer and Schiff, Siemerling, Monakow). Whether Westphal-Edinger's group belongs to this nucleus is doubted by many authors.

It is probable that the different cell-groups are centres for different muscles innervated by the oculomotor nerve, though their arrangement has not been satisfactorily determined. Hensen and Voelkers have given the following arrangement, based upon experiments on dogs: anterior cell-group for the ciliary muscle and the sphincter iridis; back of this, one for the internal rectus; then, in order, for the superior rectus, levator palpebræ superioris, inferior rectus, and superior oblique. According to Kahler and Pick, the pupillary fibres are found in the anterior root-bundle; in the posterior one, those for the external ocular muscles, the median ones being below those for the internal and inferior recti.

FIG. 203.



Frontal section through the pregeminum at the height of the oculomotor nucleus.

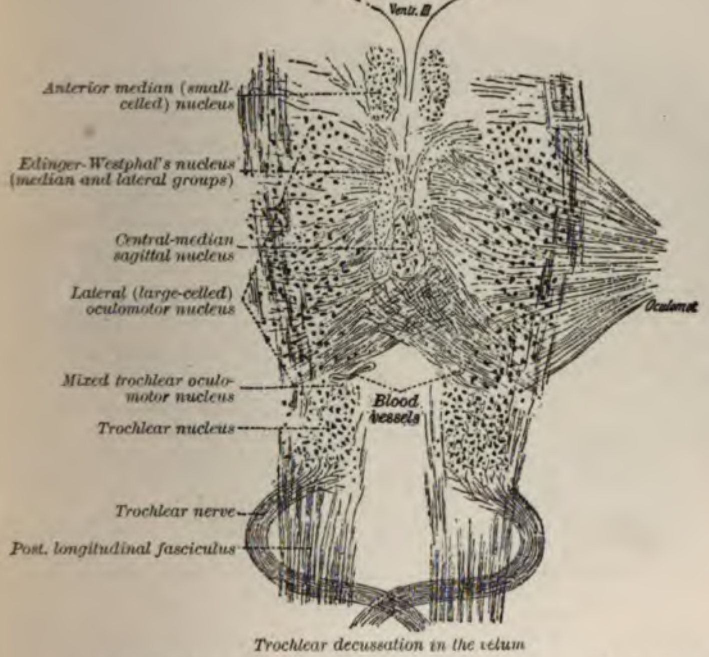
Recently attempts have been made, by means of Nissl's method, to ascertain the cell-grouping for the individual ocular muscles. According to Bach, removal of the iris and ciliary body does not cause any alterations of the oculomotor cell-area, but of the ciliary ganglion. Bernheimer has made some very complete investigations. He considers that the most anterior part of the lateral chief nucleus is the centre for the palpebræ superioris; the Edinger-Westphal nucleus, for the iris; and the median nucleus, for the ciliary muscle. Monakow does not regard it as being impossible that the cell-groups for the internal muscles of the eye may be scattered, and that only the corresponding root-fibres are found in the anterior fasciculi. He believes that the internal rectus has a double nuclear region, one in the posterior part of the dorsal nucleus of the opposite side, and a second for movements of convergence in the single central nucleus.

He reasons from the existing observations on human beings that a lesion of the oculomotor root-bundles in the posterior part of the red nucleus also involves those for the levator palpebræ superioris and internal rectus of the same side.

A very noteworthy fact is that the oculomotor in man also arises partly from the cell-groups of the opposite side (Gudden, Perlia, Bernheimer, Zappert).

This is the least true for the distal root-bundles which arise in the dorsal chief nucleus of the opposite side. Pathologico-anatomic observations also favor this idea.

FIG. 204.


Ventr. III
Anterior median (small-celled) nucleus
Edinger-Westphal's nucleus (median and lateral groups)
Central-median sagittal nucleus
Lateral (large-celled) oculomotor nucleus
Oculomot.
Mixed trochlear oculomotor nucleus
Blood vessels
Trochlear nucleus
Trochlear nerve
Post. longitudinal fasciculus
Trochlear decussation in the velum

Grouping of the nuclei of the oculomotor region. Schematic horizontal section. (After Siemerling.)

The root-fibres of the third leave the nucleus on the ventral side, traverse for the most part the corpus rotundum, and join the trunk between the cerebral peduncles.

The oculomotor nucleus is connected with the optic nerve. The tracts, however, have not been closely followed.

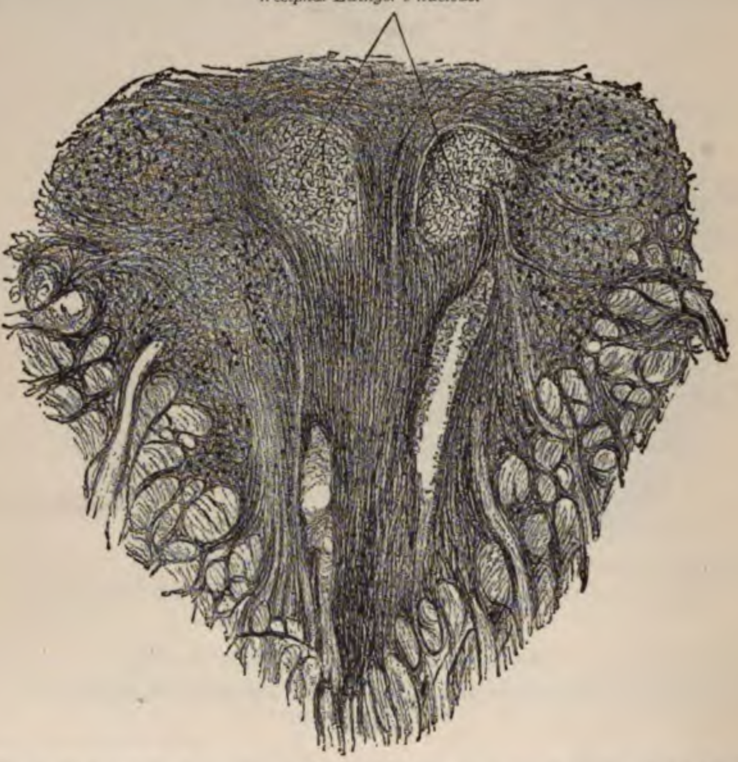
Kölliker thinks—with Meynert and others—that the optic fibres which terminate in the pregeminum act upon its cells, and these, through their nerve-processes, act, partly directly, and partly through collaterals, upon the cell-groups of the oculomotor.

Others think that the fasciculi for the pupillary innervation leaves the optic tract before this. Most investigators consider, with Gudden, that there are special fibres in the optic nerve for the pupillary reflex, that they decussate partially in the chiasm, so that the optic tract contains pupillary fibres for both eyes. Investigators differ widely as to the further course of these fibres, and their views will, therefore, not be discussed.

Reflex pupillary rigidity may perhaps be produced by lesion of the fibres connecting the terminal nucleus of the optic nerve and the nuclei of the oculomotor nerve.

Experimental observations of Gudden and Bechterew have shown that lesions of the floor and sides of the third ventricle may produce reflex pupillary rigidity. I found a degeneration of Westphal-Edinger's nucleus in a case of reflex pupillary rigidity.

FIG. 205.



Frontal section through the oculomotor nucleus in its most anterior part. Atrophy of Westphal-Edinger's nucleus. (After a carmine preparation.)

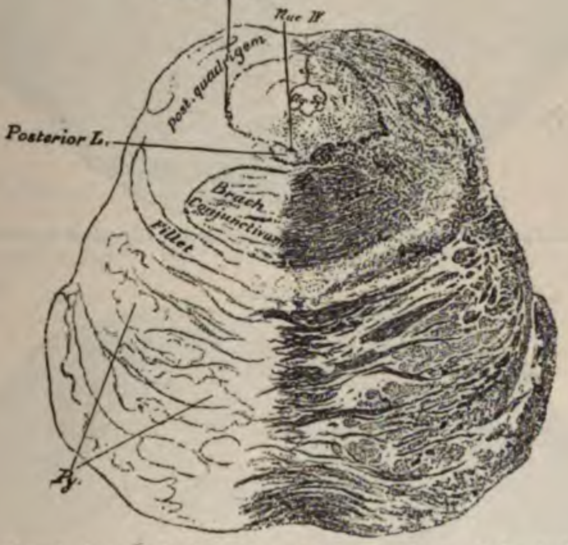
Mendel has found from experiments, that the ocular facial arises in the distal part of the nuclear area of the oculomotor nerve. Spitzka, Tooth-Turner, and others agree with him. The investigations of Schiff and Cassirer, Siemerling, and others, however, contradict this.

The posterior longitudinal fasciculus, according to many, connects the different nuclei of the ocular muscles; perhaps, also, the abducens nucleus of one side with the nuclear area of the internal rectus of the other.

Bechterew's experiments on the new-born confirm this. It probably also contains fibres which connect the trigeminal with the nuclei of the ocular nerve (Mahaim) and perhaps other fibres.

Until recently, the views as to the position of the *trochlear nucleus* differed greatly (Westphal, Siemerling, Kausch, Pacetti, Boedeker, and others). Recently, however, it has been generally accepted that it lies dorsad to the dorsal longitudinal bundle, occupying a cavity in this fasciculus. (Figs. 206–208.) Its root-fibres emerge just caudad to the postgeminum. The roots decussate completely in the medullary velum.

FIG. 206.



Frontal section through the pons in the region of the postgeminum. *L.* longitudinal fasciculus (Figs. 206–214 are stained by Weigert's or Pal's method.)

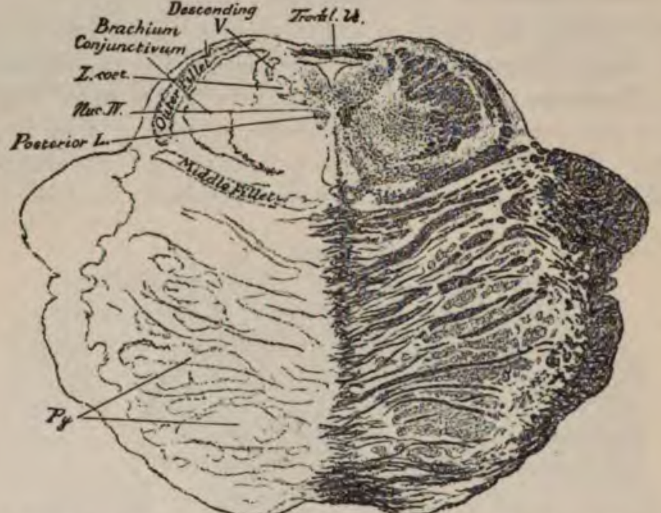
(Fig. 207.) Siemerling and Boedeker have described commissural fibres between the two nuclei.

The nuclear region of the *trigeminal* covers an extensive area.

The sensory root of the nerve arises in the *Gasserian ganglion;* from here they pass to the pons, ramifying in the sensory nucleus of the preoblongata, some of the fibres passing downward to form the spinal trigeminal root, and others ascending.

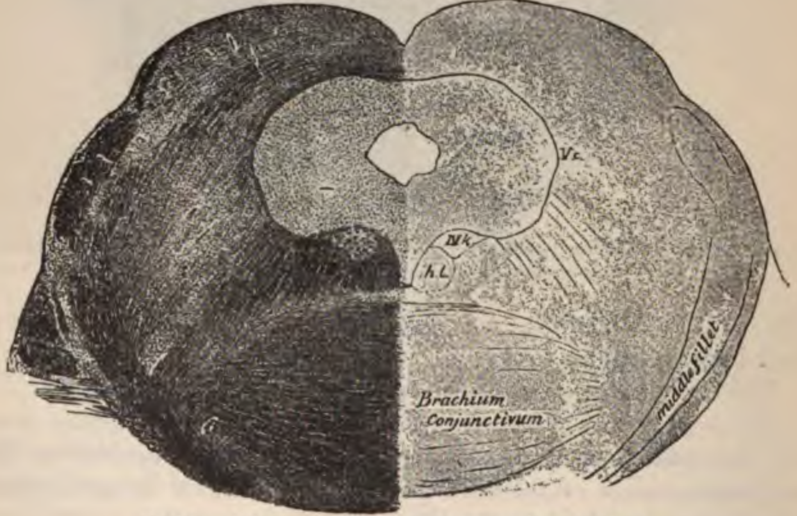
It gives off collaterals everywhere which ramify in the gray matter accompanying it (substantia gelatinosa, which finally passes over into the posterior horn of the spinal cord), and which surround the cells of this part. Collaterals are also said to pass to the facial nucleus, probably also to other motor cranial nerves. The so-called sensory nucleus in the pons is, according to Kölliker, only the beginning of the gray matter.

FIG. 207.



Frontal section through the pons at the height of the trochlear decussation.

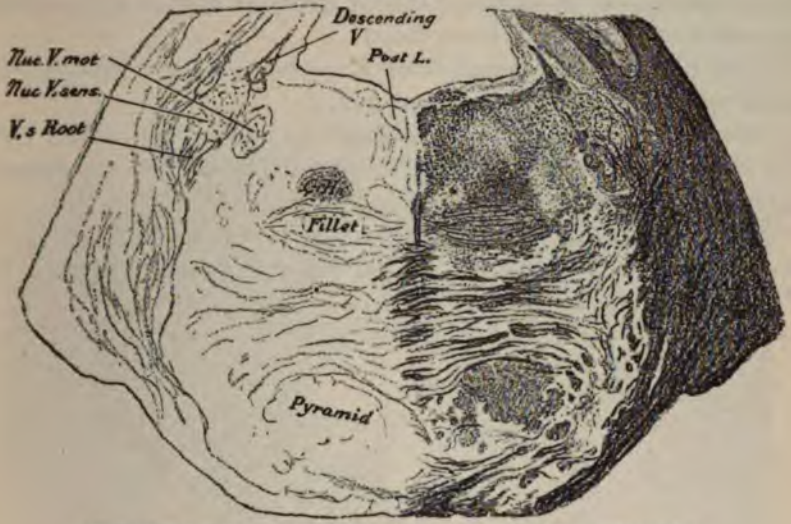
FIG. 208.



Position of the trochlear nucleus. *IVk*, trochlear nucleus.

Recent observations seem to indicate that the proximal section of this root corresponds to the third branch of the trigeminal nerve; the distal, to the first (Wallenberg, Bregmann). After disturbance of the latter, Wallenberg observed the corneal reflex disappear in guinea-pigs.

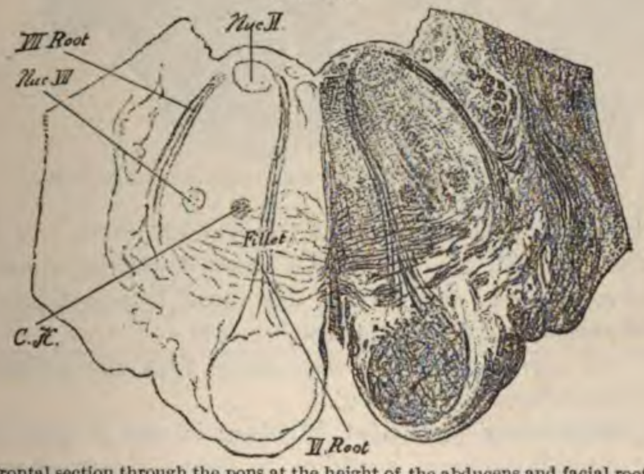
FIG. 209.



Frontal section through the pons at the height of the trigeminal nucleus.

The smaller *motor root* arises from the large-cell *motor nucleus.* (Fig. 209.)

FIG. 210.



Frontal section through the pons at the height of the abducens and facial root.

There is also a cerebral (also called the descending) root of this nerve—the mesencephalic root—which, in the shape of a small halfmoon, lies laterad to the aqueduct of Sylvius, and can be traced into the pregeminum.

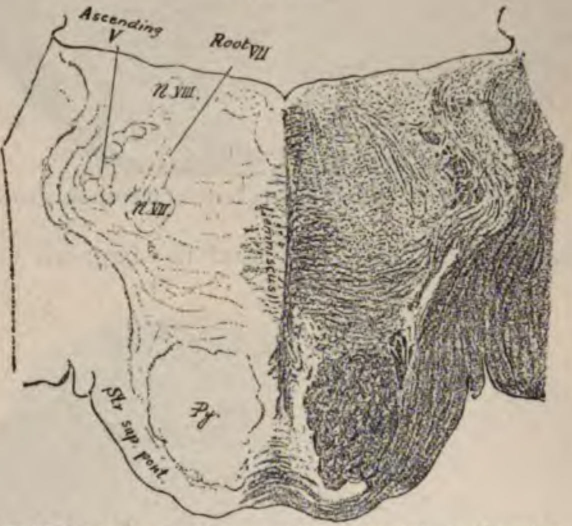
It arises in ganglion cells which accompany the root throughout its entire course. At the height of both nuclei, it reaches the emerging trigeminal root, passing between them.

This root has been regarded as a trophic one; but most investigators now consider it to be a motor root.

The position of the *abducens nucleus* may be seen in Fig. 210. The root fibres pass without decussation through the tegmentum and pyramid to the emerging nerves.

The facial nucleus (Fig. 211) lies mesad to the spinal trigeminal in the lower part of the pons and is about four millimetres in length. The roots arising in it join at the floor of the ventricle, curve over the abducens nidus from behind, and, after a short descending course dorsalward, curve obliquely outward and downward to pass finally between the facial nucleus and trigeminal root.

FIG. 211.



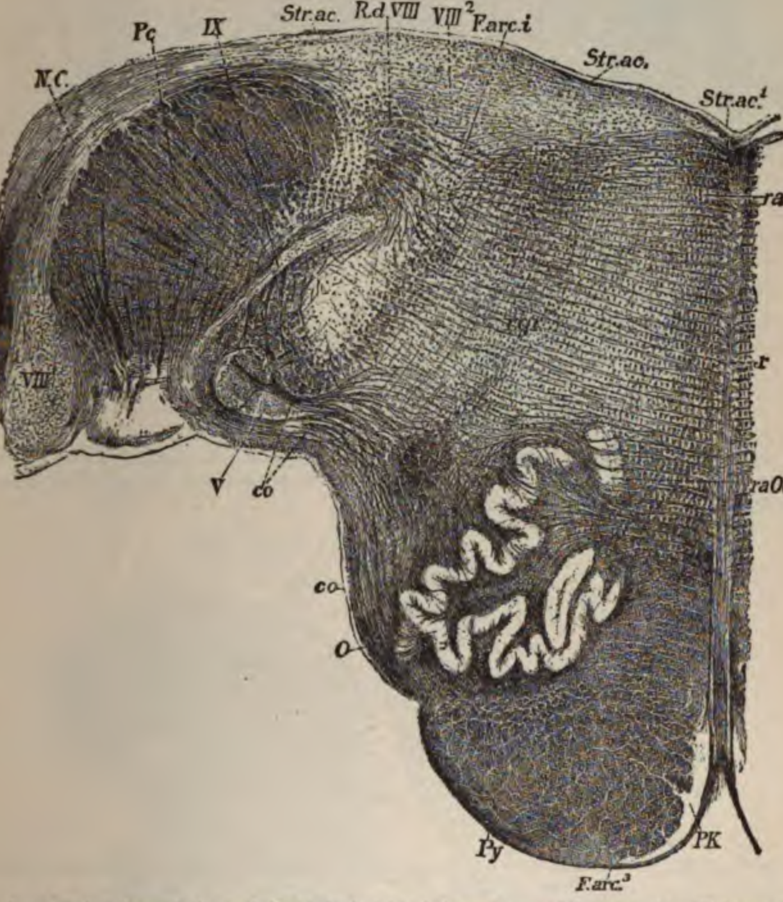
Frontal section through the pons at the height of the facial nucleus.

The root also appears to receive fibres from the nucleus of the opposite side (Obersteiner, Flatau).

The intermediary portion of Wrisberg is said to arise from the ganglion geniculi; it forms, according to Duval, the upper part of the sensory glossopharyngeal root. The chorda tympani is said to be joined to the glossopharyngeal by this nerve or pass into the solitary fasciculus.

The *acoustic* nerve consists of two roots, the *cochlear* nerve and the *vestibular* nerve. The first, probably the real auditory nerve, is said to commence in the spiral ganglion of the cochlea, and forms the external (posterior) root (Fig. 212, *N.C.*). It lies laterad to the restiform body, and terminates in the accessorius or ventral acoustic nucleus and in the acoustic tubercle.

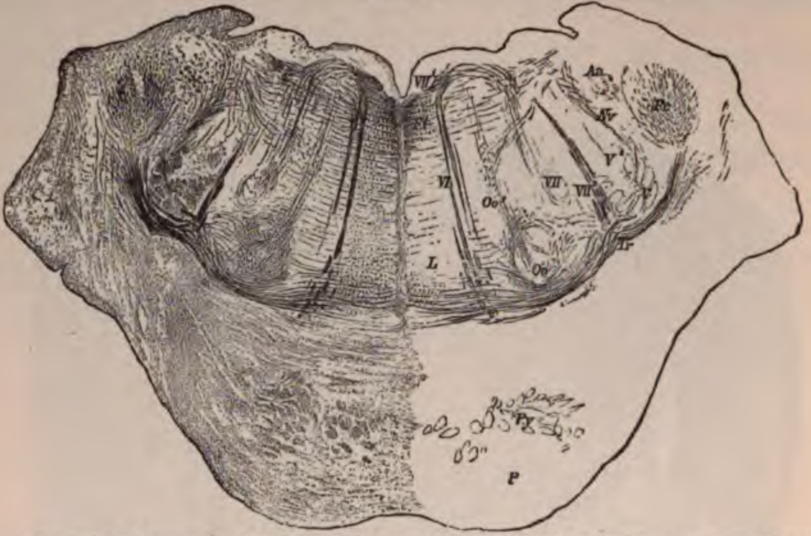
FIG. 212.



Cross-section through the medulla at the height of the entrance of the acoustic. (Enlargement 7:1.) *VIII*¹, ventral acoustic nucleus; *VIII*², dorsal acoustic nucleus; *N.C.*, cochlear nerve; *Pc*, cerebellar peduncle (restiform body); *R.d. VIII*, descending acoustic root; *Str.ac.*, acoustic stria; *co*, cerebello-olivary fibres, etc. (After Kölliker.)

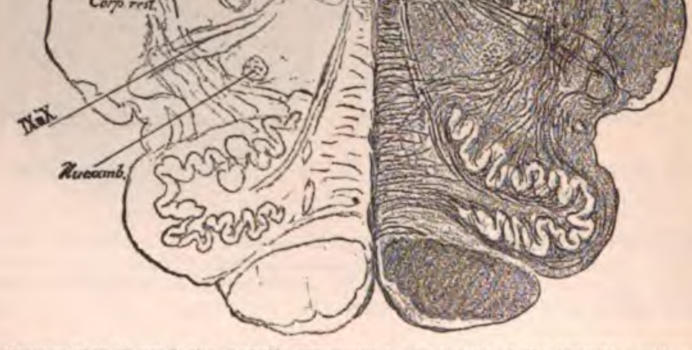
The *vestibular nerve*, the anterior mesial root, arises in the labyrinth (ganglion scarpæ). It probably has no auditory functions, but conducts impressions which influence co-ordination to the central organs, particularly to the cerebellum. It ends partly in the dorsal mesial acoustic nucleus (chief nucleus) (Fig. 212), partly in Deiters's nucleus (Monakow), and in its lateral and cerebellad continuation, the vestibular nucleus (Bechterew).

FIG. 213.



Cross-section through the distal part of the pons of an eight-months embryo. *Oo*, upper olivary body; *Tr*, trapezium; *L*, fillet; *Nv*, vestibular nerve; *VII'*, facial knee. Pyramids and transverse pontile fibres still unmedullated. (After Kölliker.)

FIG. 214.



Cross-section through the medulla oblongata at the height of the acoustic nucleus.

From here fibres pass to the cerebellum, which are said to transmit impulses to the co-ordinating centres from the labyrinth.

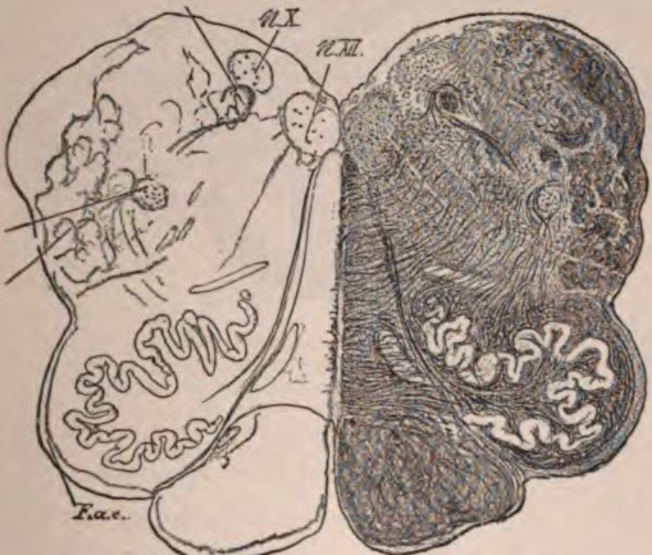
Externally to the dorsal acoustic nucleus lies a tract passing spinalward, and which is a continuation of the funiculus cuneatus of the spinal cord, which is perhaps a descending acoustic root (Roller). The vestibular nerve seems to be continued in it.

The *acoustic striæ* arise for the most part from the acoustic tubercle. They appear to form a sensory tract of the second order, and after decussation in the raphe probably pass to the lateral lemniscus.

The vagus and glossopharyngeus cannot be sharply separated from each other; particularly is this true of the intramedullary roots.

The sensory vago-glossopharyngeal arises, according to our present knowledge, in ganglia which are situated outside of the medulla (jugular, petrosal, etc.). It enters the medulla oblongata, and forms here the solitary bundle or fasciculus, formerly called the ascending vagus root, but which in reality is a descending sensory vago-glossopharyngeal root. (Its position can be seen in Figs. 214, 215, and 200.)

FIG. 215.



Frontal section through the medulla oblongata. (Weigert's stain.)

The fibres give off collaterals at all heights, which ramify in the gray matter accompanying it. Another part of the sensory root of the vagus and glossopharyngeus passes to the posterior vagus nucleus on the floor of the fourth ventricle.

The nucleus ambiguus is probably a motor nucleus of the vago-glossopharyngeus.

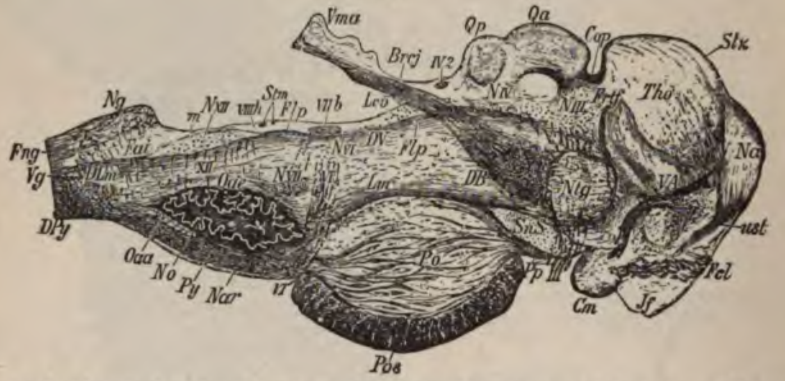
A small large-cell nucleus is found in the proximal part of the solitary bundle. This may be a special motor nucleus for the glossopharyngeus.

Fibres from the terminal nucleus and the cells of the solitary bundle are said to pass to the lemniscus of the opposite side.

Consult pages 115 and 116 for the position of the nucleus accessorius.

The *hypoglossal* nucleus (Figs. 214 and 215) lies close against the raphe. It is supplied with large cells and a rich net-work; its roots leave the medulla between the oliva and pyramid. The cell-group lying ventrad to the twelfth nucleus has probably nothing to do with the hypoglossal.

FIG. 216.



Combined sagittal section through the brain-stem (Obersteiner). (Pal's stain.) *III*, oculomotor nerve; *IV2*, decussation of the trochlear nerve; *VI*, abducens nerve; *VIIb*, ascending facial limb; *XII*, hypoglossal nerve; *Brcj*, brachium conjunctivum; *DB*, decussation of the brachium conjunctivum; *Fai*, internal arcuate fibres; *Fcl*, columna fornicis; *Flp*, posterior longitudinal fasciculus; *Lm*, lemniscus; *No*, oliva; *Niii*, oculomotor nucleus; *Niv*, trochlear nucleus; *Nvi*, abducens nucleus, etc.; *Po*, pons; *Pp*, pes pedunculi; *Py*, pyramids; *Fng*, funiculis gracilis; *Tho*, thalamus opticus; *Qa*, *Qp*, anterior and posterior quadrigemina; *Ntg*, red nucleus.

Fig. 216 will also give an idea of the position of the nuclei and the course of the nerve-roots, and Fig. 217 the places of exit at the base of the brain.

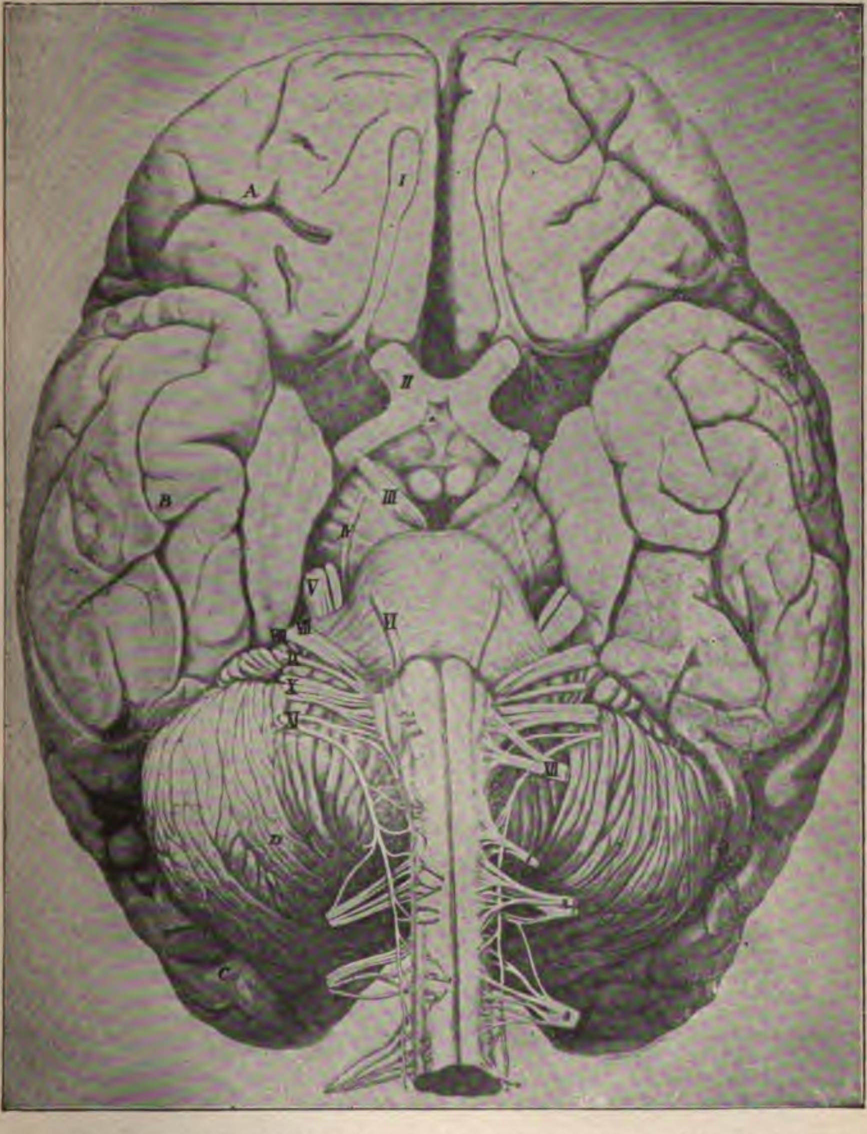
A. Westphal has determined the following facts concerning the development of the cranial nerves: At birth the motor nerves are medullated; the sensory and mixed ones, excluding the acoustic, are not. The optic nerve is the last to develop. In the third week the formation of nerve-sheaths can be noticed.

THE BLOOD-SUPPLY OF THE BRAIN.

All arteries of the brain come from the *carotid* and *vertebral* arteries.

The vertebral arteries coalesce to form the basilar artery, and this in turn divides into the posterior cerebral arteries. The anterior cerebral and the anterior communicating arteries in front, the internal carotid and posterior communicating and the posterior cerebral behind, form the circle of Willis, which surrounds the chiasm, tuber cinereum, and corpora mammillaria.

FIG 217.



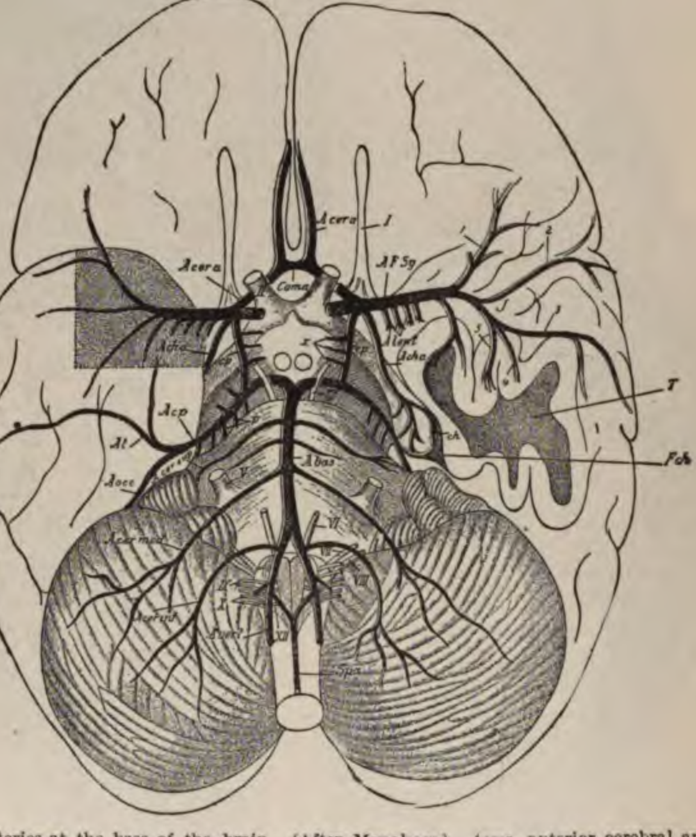
Base of the brain. Origin of the cranial nerves. They are numbered in Roman letters. *A*, frontal lobe; *B*, temporal lobe; *C*, occipital lobe; *D*, cerebellum.

Branches from these arteries ramify in the cerebral substance, branching, without anastomosing, in the central ganglia and the surrounding parts.

On the rest of the upper surface of the brain the arteries branch in the pia, and from this pial arterial region, to which neighboring arteries are connected by anastomosis, short and long branches pass to the cortex, the latter to the subcortical areas. We have, therefore, a *central* and a *cortical* arterial net, which, outside of capillary communication, do not anastomose with each other.

From the *artery of the Sylvian fissure* or the median cerebral artery, the arteries for the basal ganglia and the internal capsule, with the exception of its posterior part, are given off. Those passing to the striate body are called lenticulo-striate arteries; those supplying the thalamus are known as lenticulo-optic arteries. Anastomoses between them do not occur. The choroid artery comes from the median cerebral artery, runs along the optic tract, and passes to the choroid plexus of the lower horn. It sends branches to the posterior part of the internal capsule.

FIG. 218.



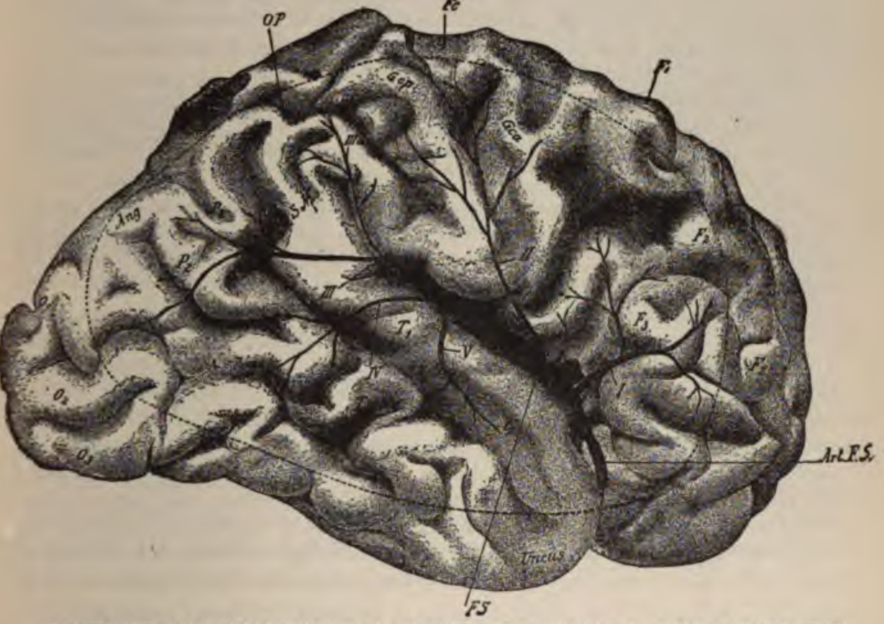
The arteries at the base of the brain. (After Monakow.) *Acera*, anterior cerebral artery: *Coma*, anterior communicating artery; *AFSy*, artery of the Sylvian fissure; *Alent*, lenticular artery; 1–5, the cortical chief branches of the Sylvian artery; *cp*, posterior communicating artery; *Acha*, anterior choroid artery; *x*, lateral branches of the posterior communicating artery; *Acp*, posterior cerebral artery; *Abas*, basilar artery; *At*, temporal artery; *Aocc*, occipital artery; *A cer sup*, superior cerebellar artery; *A cer med*, median cerebellar artery; *A cer inf*, inferior cerebellar artery; *Spa*, anterior spinal artery.

The *posterior cerebral artery* supplies the occipital lobes (for the most part), the posterior part of the optic thalamus; it also sends branches to the tegmentum, the cerebral crus, the gemina, and the nucleus of the oculomotor nerve.

The anterior cell-groups are supplied by a special arterial branch. The inner arteries of the peduncle and oculomotor nucleus are terminal arterioles. The posterior cerebral artery supplies, therefore, both the visual and ocular muscle centres. It sends the occipital artery to the occipital lobe, from which arise the parieto-occipital, calcarine, and cunei arteries.

The *anterior cerebral artery* supplies the frontal lobe, with the exception of the third frontal convolution, and the cortex of the mesial wall of the hemisphere as far as the precuneus, and also the corpus callosum.

FIG. 219.



Lateral view of the right hemisphere showing the area supplied by the middle cerebral artery. (After Monakow.) *Art.F.S.*, artery of the Sylvian fossa; *I–V*, the five chief branches of this artery; *Fc*, central fissure; *FS*, Sylvian fossa; *OP.*, interparietal fissure; *t*, first temporal fissure; F_1–F_3, first, second, and third frontal convolutions; *SM*, supramarginal gyrus; *Ang*, angular gyrus; O_1–O_3, first, second, and third occipital convolutions. The dotted line shows the area supplied by the Sylvian artery.

The middle cerebral artery spreads over the island of Reil in four or five branches (Fig. 219). The first supplies the third frontal convolution; the second, the facial and arm centres on the anterior central convolution; the third, the posterior central convolution and the parietal lobes; the fourth, the lower parietal lobule and, with the fifth, the upper temporal convolutions.

The pons, medulla oblongata, and cerebellum are supplied by the vertebral and basilar arteries. Their branches are terminal arteries.

The arteries of the pons and oblongata consist of the median or nuclear arteries and the radicular arteries. The first approach the nuclei of the nerves in the median line, the latter run alongside the nerve-roots and give off a branch which accompanies them to the periphery, and another, which enters the nucleus. The branches for the facial and acoustic may come from the vertebral or from the basilar, or from both. The other radicular arteries arise in the vertebral or basilar, or from both.

The nerve-roots coming from the medulla oblongata are supplied by the vertebral artery, with the exception of the hypoglossus, which is supplied by the anterior spinal artery. The left vertebral is, as a rule, wider than the right. The branches for the olives and pyramids arise from the vertebral or anterior spinal arteries; those for the restiform body from the inferior cerebellar artery, which also sends branches to the roots of the vagus, accessorius, spinal trigeminal root, and the reticular field.

SECONDARY DEGENERATION IN THE CEREBRUM.

The same laws as have been developed for the spinal cord govern here also. Nerve-processes separated from their cells of origin degenerate rapidly. Disturbances of the motor region, therefore, produce a degeneration of the whole conducting tract coming from it. All the fibres distad to the disease-process degenerate, no matter where the lesion occurs in the motor paths.

Secondary alterations of the nerve-cells separated from their nerve-processes also occur in the brain. This is especially true of diseases occurring in early childhood. Monakow cites degeneration of the giant pyramidal cells connected with the peduncle after section of the peduncle in young animals.

More exact conditions for the occurrence of cellulipetal degeneration are not yet known. It is certain that all cells need not degenerate. The nearer to the cell the division of the axis-cylinder occurs, the more it is influenced and injured (Forel), as the collaterals would then also be involved.

Monakow characterizes as *secondary atrophy of the second order* alterations which do not consist in gross degeneration, but simply in decrease in volume. Here belong, for example, atrophy of the lemniscus after extensive, early acquired defects in the parietal lobes of the brain, atrophy of the ocular nerves after old lesions in the occipital lobe, etc. After some years a retrogressive metamorphosis and complete atrophy may occur (Henschen). The cortical lemniscus may atrophy after lesions in the central convolutions, but only then when the lesion is extensive, when almost the whole motor hemisphere and the parietal brain are disturbed, and the lesion has been early acquired (Monakow). Old lesions which disturb the tegmentum of one side produce ascending and descending degeneration of the fillet. The ascending degeneration, according to Dejerine, Monakow, and Schlesinger, does not extend farther than the optic thalamus.

GENERAL SYMPTOMATOLOGY OF CEREBRAL DISEASES.

I. GENERAL PHENOMENA.

Only the organic diseases of the brain will be considered at this place; the functional diseases will be discussed in another section.

The symptoms of cerebral diseases are determined less by the nature of the process than by the location of the disease.

Certain symptoms occur in diseases of all cerebral areas. We call these *general cerebral symptoms*, in contradistinction to *focal symptoms*,—*i.e.*, those produced by lesion of a particular part of the brain.

The diagnosis—cerebral disease—is based to a great extent upon the general symptoms.

The nature of the anatomic process is also to a great extent revealed by these symptoms. Focal symptoms show the location of the disease, or help us to localize it.

We include among the general symptoms, *headache, vomiting, vertigo, disorders of consciousness* and *psychic defects, and alterations of the pulse, respiration, temperature,* etc. The spasms, which are more often a focal symptom, will be considered at another place.

Choked disk, the most important symptom of increased intracranial pressure, will be discussed under diseases of the optic nerve.

HEADACHE is one of the most constant symptoms of cerebral disease. Its diagnostic importance is, however, lessened from its occurring also in functional cerebral disorders and also in affections of other organs without it being possible to differentiate it sharply from headache produced by other diseases.

Its association with other cerebral diseases is necessary to enable it to aid in making a diagnosis. In general, we might say that a particularly intensive and persistent headache is produced by an organic cerebral disease. Even this does not mean much, as hysteria and neurasthenia may be accompanied by a persistently severe headache, and such a form of cephalalgia occurs also in migraine or modifications of it. Headaches in hysteria and neurasthenia are generally dependent upon psychic influences and upon an abnormally increased self-observation. For other differential points consult the appropriate chapters.

The headache caused by brain diseases is generally increased by coughing, sneezing, etc. This is, however, also the case in headache caused by circulatory disorders, especially venous stasis. Many forms of neurasthenic headache also possess this characteristic. The headache accompanying cerebral diseases is either diffuse or localized. In the latter case, the region of the headache may correspond to the seat of the lesion, though just the opposite also occurs.

The combination of headache and VOMITING indicates an organic cerebral disease, provided fever, intoxication, uremia, migraine, and gastric disorder are excluded. The vomiting occurs, as a rule, at the height of the cephalalgic attack. The ease with which it occurs is also characteristic. Neither gastric pains nor choking movements precede it, and, often, not even a long-continued stage of distress, but the gastric contents are ejected easily and suddenly. The vomiting occurs independently of the ingestion of food,—for example, on an empty stomach; it may, however, occur after eating. Change of position, as elevation of the head, may induce it. Though this projectile vomiting may occur in diseases of every part of the brain, it is specially marked in affections of the cerebellum and medulla oblongata.

VERTIGO is a symptom of minor diagnostic importance. Although various sensations are associated with this name, as a rule, it refers to a *sensation of incoördination* of sudden onset, which disappears suddenly or gradually. The patient feels as if the floor moved under him, as if everything turned around him, or as if his own body were rotating. A fleeting disturbance of consciousness is also characterized, by patients, as vertigo. This symptom may be due to various causes (see chapter on vertigo), so that it is only of diagnostic importance when combined with other symptoms. A particularly severe and stubborn form of vertigo occurs in diseases of the cerebellum and cerebellar peduncle, and is characterized here by objective signs of incoördination. A similar form of vertigo is produced by labyrinthine disease.

DISORDERS OF CONSCIOUSNESS; PSYCHIC DEFECTS.—Psychic disorders occur quite often in organic cerebral diseases. They are produced by diffuse, rather than by circumscribed, affections.

Consciousness may be disturbed in many ways. According to the severity, we distinguish *hebetude*, *somnolence*, *sopor*, and *coma*. When somnolent the patient is sleepy, but can be easily awakened, soon to fall again into a light sleep or doze. In sopor the patient can be awakened only by strong sensory stimulation. In coma there is complete unconsciousness, with annulment of the reflexes, and the patient cannot be awakened.

It is difficult to recognize slight degrees of stupor. They are often thought to be conditions of imbecility. Notice that the patient when awake thinks clearly and reasons correctly, but that it is difficult for him to concentrate his mind upon the matter; he looks vacantly into space and pays little attention to what is going on around him. A noteworthy sign of impaired consciousness is the fact that urine and feces are passed involuntarily, though the sphincters are normal. In the severe forms, the patient can forget to chew and to swallow, and retains his food a long time in his mouth. In coma it cannot be swallowed at all.

A severe grade of clouded consciousness in the wakeful state, in which the external world and its doings seem to be entirely lost to the patient, may also be called *stupor*. The reflexes are present in this condition.

Chronic cerebral diseases, which extend over a large part of the brain, or, through pressure, involve the whole brain (tumors), may produce a gradually increasing disturbance of consciousness, which finally passes over into unconsciousness. A suddenly occurring unconsciousness is caused by cerebral hemorrhage or by vascular obstruction, also by traumata, and even by severe psychic emotion. The loss of consciousness in epileptic attacks also occurs suddenly. When produced by loss of blood (cerebral anemia) and intoxication, it generally comes on gradually.

Complete unconsciousness of sudden onset is called *apoplexy*, though it has become more and more the custom to call coma, produced by cerebral hemorrhage, by this name. As an embolus of a cerebral artery may produce the same condition, the word apoplexy does not convey the cause of the attack.

It is rare for the cerebral hemorrhage to produce a disturbance of consciousness which gradually increases to coma (ingravescent apoplexy). The specific signs of an apoplectic stroke are described in the chapter on cerebral hemorrhage.

Delirium is a characteristic form of disturbed consciousness—a condition of mental excitation and confusion, with hallucinations and motor restlessness. Delirium, however, more rarely results from organic cerebral diseases than from *fever*, *intoxication* (alcoholism, morphinomania, autointoxication in diabetes, carcinoma, etc.), and *infection*.

A special form of delirium is that in which the patient, controlled by his hallucinations, murmurs and mumbles continually, constantly moving his hands at the same time, as if he desired to grasp or tear something, etc. In other cases the delirium is combined with marked forcible movements, so that the patient cannot be kept in bed, seeks to jump out of the window, etc.

A not uncommon sign of organic brain disease is decrease or loss of mental strength, from simple *weakness of the memory* extending to complete *dementia*. These disorders are produced particularly by chronic, diffuse, and disseminated processes which involve the cerebral cortex directly or through the blood-vessels.

The memory may be disturbed in various ways. New memory-pictures may not be taken up, or the memory for a certain period is impaired, or the memory for names only may be lost, etc.

PULSE, RESPIRATION, AND TEMPERATURE.—The pulse is retarded, accelerated, or irregular. Cardiac disorder occurs especially in disease of the medulla oblongata or of the vagus centre involving it directly or from increased intracranial pressure. The characteristic symptom is the slowing of the pulse, which may sink to from twenty to thirty beats a minute or lower (tumor cerebri, meningitis, abscess, etc.), though it rarely goes below from forty-four to forty-eight beats. An acceleration of the pulse may follow the retardation in the last stages. An acceleration may also be the first sign of a vagus affection. The usual acceleration observed in fever may be absent in cerebral disease; the pulse may even be slow with a high temperature.

Irregularity occurs especially in diseases of the medulla, or in those which involve the vagus centre secondarily.

The *respiration* may also be influenced by cerebral disease. In coma and in increased intracranial pressure, the breathing becomes less frequent and deeper. This also occurs in diseases of the medulla oblongata. Dyspnea and irregular breathing, however, occur more often in this condition. A particular form of this is *Cheyne-Stokes breathing*, which occurs in meningitis, hemorrhages, tumors, aneurisms of the vertebral artery, etc., and which may be present in coma also. It is an arrhythmical breathing of a periodic type. A few superficial breaths are succeeded by gradually increasing rapid and deep breaths, finally becoming noisy and snarling. They gradually decrease in rapidity and depth; then comes a stage of apnea, following which the cycle commences anew. During the apnea, the pupils become smaller and the pulse slower. Traube regards as a cause of this phenomenon the lessened excitability of the respiratory centre; according to Filehne, the vasomotor centre has much to do with it. A similar type of breathing may occur in healthy individuals during sleep.

Biot's breathing is a short, rapid breathing, of sudden onset and interrupted by pauses of a half-minute or so.

There is a form of Cheyne-Stokes breathing in which the period of apnea is absent.

Kussowitz describes, as another form of respiratory disturbance, an expiratory apnea; and Schlesinger describes, as a type allied to this, the following: after a deep inspiration, rapid expiratory impulses occur without any inspirations between; the thorax finally remains fixed in the position of expiration, and the attack closes with a deep inspiration.

A simple irregularity of the breathing occurs in diseases of the medulla. In rare cases cessation of breathing with continued cardiac action has been observed, so that artificial respiration prevented death.

The temperature is usually increased in infectious diseases located in the brain (meningitis, encephalitis, etc.), but may not be elevated. The fever of cerebral disease may be accompanied by slowing of the pulse. Cerebral hemorrhage generally causes a slight increase of the temperature, but rarely high fever. The apoplectic attacks of sclerosis and paralytic dementia are generally combined with fever. A steady increase of the temperature is observed in the status epilepticus. Every disease of the pons and medulla may cause fever, especially acute and destructive processes. Acute diseases of the motor zone and the striate body cause an increase of temperature. Decreased temperature is not a rare symptom in cerebral hemorrhage, and also occasionally occurs in cerebral abscess. It is occasionally observed in comatose conditions of a different origin.

II. FOCAL SYMPTOMS.

MOTOR FOCAL SYMPTOMS.

A. Symptoms of Irritation.—Irritation of the cortical motor zone produces convulsions in the musculature of the opposite side of the body, a fact which was first proven by Fritsch and Hitzig. Disease-processes which involve the motor cortical area, without injuring it, cause tonic and clonic spasms in the muscles whose centres have been irritated. A tonic spasm generally precedes the clonic twitching. These motor phenomena of irritation may be confined to a muscular group or to one extremity. The twitchings excited by repeated or severe irritation do not limit themselves to the muscular group first involved, but extend over the whole side of the body, as if the irritation in the brain had extended to neighboring centres. If, for example, the disease is in the facial centre, it may commence with twitchings of the facial muscles. In its further course, when the irritation gradually increases or is intense from the beginning, the convulsions also begin in the facial area, but extend then to the arm (hand and fingers first) and finally to the leg of the same side. If they commence in the leg, they extend to the arm and, finally, to the face. If they start in the arm, they generally next involve the face, and next the leg of the same side.

Consciousness generally remains intact in local muscular spasms; it may, however, disappear in the course of the spasms if they extend over the entire half of the body.

Unconsciousness also generally occurs when the other side of the body becomes affected. In passing over to the other side the spasm involves first the muscular area which last twitched or the region which was first attacked by the spasm on the other side. The views expressed are not unanimous on this point.

Those muscles which normally act bilaterally, as the truncal, maxillary, laryngeal, pharyngeal, and ocular muscles, may, in unilateral spasms, be also affected bilaterally. I have noticed this particularly in regard to the orbicularis palpebræ.

This form of localized or *unilateral spasms* is called *partial*, *cortical*, or *Jacksonian epilepsy*. It is caused by any irritative condition of the cortical motor zone. It may be of *functional* origin (hysteria, etc.), or *organic* (hemorrhage, softening, inflammation, especially neoplasms), or caused by *intoxication* (alcoholism, uremia, lead poisoning, etc.).

A condition of *temporary paralysis* generally follows the spasms, the muscles which were first and most affected by the spasms being principally involved. This is probably an indication of exhaustion of the motor centres following the irritation, and soon disappears.

The nature of the cause of the irritation often, however, is such that the cortical centres are not only irritated, but also to some extent injured. This is the reason that chronic conditions of paralysis are often observed in cortical epilepsy. The paralysis may occur from the beginning in acute destructive affections (hemorrhage, softening, etc.), even precede the spasms; while in chronic progressive forms it comes on gradually or in step-like gradations. The lesion which excites the spasm need not directly involve the motor cortex, but may be only near enough to irritate it. A disease, however, which produces paralysis of the cortical centres must have its seat in this region, or impair its functions by pressure.

If a local spasm appears first, which only extends farther in later attacks, and if the paralysis spreads in a similar manner, it indicates a slowly progressive process which is always of an organic nature.

True epilepsy rarely commences with local muscular twitchings; it also rarely limits itself to one side of the body.

Partial epilepsy may also confine itself to the *sensory* area, or affect both motor and sensory functions together. Paresthesia in a limb or a part of a limb may usher in a spasm, accompany the twitchings, or be the only symptom of irritation,—*i.e.*, to a certain extent, an equivalent of the attack. The local diagnostic significance of these attacks is not positively established, but we know that they always occur in diseases of the motor cortex. Tachycardia has also been noticed accompanying cortical epileptic attacks. Hemilateral twitchings may also be excited by disease of the *subcortical* medulla, especially when they involve the conducting tracts coming from the motor zone. They do not entirely correspond to the Jacksonian type, and when very similar, show that the cortex is in some way involved (pressure, etc.). With the complete destruction of the cortical centres, the cortical epilepsy disappears.

Diseases which cause an increase in intracranial pressure may also cause convulsions. The spasms produced by general increase of cerebral pressure are, however, *general* and resemble, to a lesser or greater extent, *true epilepsy.* Clonic twitchings, lasting days and weeks, are rarely caused by diseases of the motor centres. Clonic facial spasms were observed for some time in a case of a tumor near the facial centre. I observed for days repeated rhythmical muscular twitchings in the muscles of the toes in a case of tumor of the leg centre. This form of spasm also occurs in paralytic dementia. Kemmler described attacks of spasms with rhythmical twitchings isochronous with the pulse.

It is probable, but not definitely settled, that choreic twitchings, athetosis, and similar motor phenomena of irritation come from the cortex. These phenomena will be discussed under hemiplegia.

Tetanoid spasms have been observed in diseases of the cerebellum, particularly in tumors: a paroxysmal, tetanic, muscular rigidity of the entire body, with opisthotonos, similar to that of tetanus. The tonic contraction is generally broken by general, or a succession of clonic, twitchings.

I observed continual twitching of the laryngeal muscles and of the palatine velum in a case of cerebellar tumor which pressed upon the medulla.

B. PARALYSIS.—Destructive diseases of the motor centres and conducting tracts are the cause of the paralysis developing in cerebral diseases. A cortical paralysis differs from one depending upon an affection of the conducting tracts in being confined, as a rule, to one section of one side of the body,—a monoplegia. This is not surprising when we consider that the motor centres are spread over a large cortical area, and that the organic cerebral diseases appear mostly in the form of circumscribed lesions. The vascular supply to this region is from different arteries. The monoplegia is also generally a paresis, and not a complete paralysis.

A cortical disease limited to the facial centre produces a facial monoplegia. If the lower part of the anterior central convolution is also involved, a facio-lingual monoplegia occurs. It is more common, however, for the process to involve the arm centre also, so that a faciobrachial monoplegia is the result. Or, in addition to the facial, a few muscles of the arm,—*e.g.*, those of the hand and finger, are paralyzed. The paresis may involve the thumb alone, or the fingers with the exception of the thumb. A partial radial paralysis of central origin has been observed by myself, and also by Pick. If the disease has its seat exclusively in the paracentral lobes, it may cause a pure crural monoplegia. A cortical paralysis, confined to the extensor longus hallucis, may even occur.

A brachial monoplegia is caused by a lesion of the middle third of the central convolution. If only the upper two-thirds of the central convolution were affected, we would find paralysis of the arm and leg, the cranial nerves (vii and xii) being spared. A diffuse disease of the whole motor zone will also produce hemiplegia.

Monoplegia is characteristic of cortical motor diseases. On the other hand, we have been taught by brain surgery that superficial cortical lesions which do not extend into the medulla do not entail persistent paralyses. Subcortical lesions immediately below the motor cortical gray matter may produce monoplegia. The deeper the lesion lies, the larger the number of motor fibres that are disturbed, and the form of paralysis produced, therefore, is generally a *hemiplegia.* In a few cases paralyses of a monoplegic character are also observed in diseases of the internal capsule. Paralyses of the masticatory, deglutitory, and laryngeal muscles are almost never produced by unilateral cortical lesions, but bilateral lesions of the corresponding centre may evoke a bilateral paralysis of these muscles.

Cortical epilepsy and *monoplegia* are the attributes of cortical diseases in the central motor area. The first is a sign of irritation; the latter shows loss of function, which may be produced by inhibition, intoxication, hemorrhage, softening, inflammation, abscess, etc.[1]

Monoplegia is almost always combined with *increased deep reflexes* and contracture.

The muscles withdrawn from the control of the will may, however, still act under certain conditions; as, for instance, in strong emotion, gesticulation (reflexly), and accessory movements. I treated, for example, a woman who had had for a long time a cortical monoplegia of her arm resulting from a tumor of the motor zone, and who was unable to move the arm in the least. When she was about to be placed under the influence of chloroform, however, in her excitement she commenced to make marked movements of repulsion with her paralyzed limb.

A motor disturbance has been described by Krafft-Ebbing and Bruns under the name "mental paralysis." It is probably due to the motor centres being deprived of their usual sensory stimulation. The will can still act upon the muscles, but the extremity to a certain extent does not exist any longer for the patient, because the association tracts are injured or broken. According to Bruns this condition can be recognized from the fact that the patient may use his muscles upon request and, perhaps, with full strength.

We find, in the posterior limb of the internal capsule, all the motor conducting tracts for the opposite side of the body. Diseases of this region almost always produce *hemiplegia,*—*i.e.,* paralysis of the leg, arm, facial muscles, and tongue. Only the lower facial area is, in general, involved in the facial paralysis. The mouth is drawn towards the sound side, and can only be moved in this direction. The upper facial region is spared, or only slightly impaired,—to such a degree, perhaps, that the eye on this side may not be as tightly closed as the one on the sound side, and that the orbicularis can generally only be contracted conjointly with that of the sound side. In rare cases, a slight logophthalmos may occur.

---

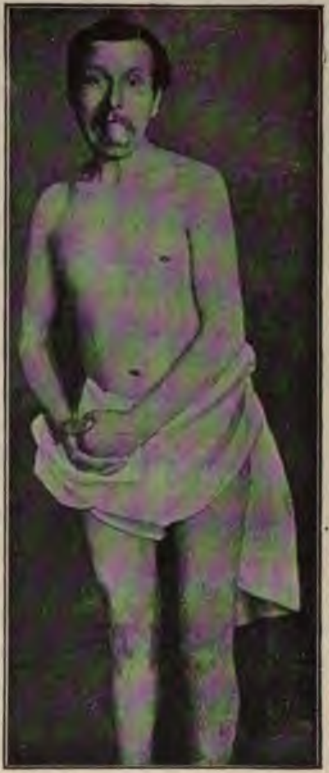
[1] Recent experimental investigations of Hernig and Scherrington show that inhibitory impulses may also arise in the cortex.

The tongue deviates to the paralyzed side, showing that the hypoglossus is involved. (Fig. 220.) Unilateral paralysis of the facial and hypoglossal nerves often produces a slight articulatory disturbance, which, as a rule, rapidly disappears again. If the hemiplegia is an incomplete one, the hand and fingers, as well as the foot and toes, are paralyzed to a greater extent than are the other muscles of the extremities. But even in those cases where the hemiplegia has developed in a typical manner, the paralysis does not affect all parts equally: the extensors of the foot and the flexors of the knee are most affected, and, as a rule, persistently paralyzed (Wernicke).

The muscles, also, which open the hand and which rotate the extremity outward tend to remain paralyzed (Mann).

The *maxillary, deglutitory, laryngeal,* and most of the *truncal* muscles—*i.e.,* those muscles which normally act bilaterally—are spared. It has been assumed that each hemisphere contains centres for the bilaterally acting muscles of both sides, so that partial lesion of one side does not produce much of a defect in this respect. This probably also explains the slight involvement of the orbicularis palpebrarum; and that the ocular muscles are never involved in simple hemiplegia is probably also due to this. The deviation of the head and eyes at times accompanying hemiplegia is a symptom of short duration, the importance and meaning of which will be discussed later.

FIG. 220.



A case of hemiplegia sinistra. Deviation of the outstretched tongue towards the left side.

Of the truncal muscles, the trapezius is generally affected. The shoulder on the paralyzed side can be only partially elevated or not at all, though the sternocleidomastoid contracts normally. The thorax, in deep respiration, expands less on the paralyzed side (Nothnagel). In bilateral lesions the deglutitory, masticatory, and larnygeal muscles are also involved (see Pseudobulbar Paralysis). The muscles of the neck and trunk were affected in a few cases of this kind.

A homolateral or collateral hemiplegia has been noticed in a few cases,—*i.e*, a unilateral paralysis on the same side as the lesion. Most of these cases were, however, diagnosed or observed wrongly, the alterations in the opposite hemisphere or in the pons or medulla which touched the motor paths at this point not having been noticed. A process arising in one hemisphere, especially a tumor, may so press upon the other side as to injure it the most. An immovability and atony of a limb occurring in coma has often falsely been regarded as hemiplegia, and the symptoms of motor excitation occurring in the other side were falsely thought to be signs of activity (Pineles, Ortner). In the few cases in which these remarks do not apply, a developmental anomaly —a congenital absence of the pyramidal decussation—has been regarded as a cause.

Those cases in which a hemiplegia is a *direct focal symptom*—*i.e.*, caused by an injury of the motor conducting tract—undergo in time certain alterations. Some muscles recover their movement. The glossal paralysis, for instance, soon disappears. The leg recovers sufficiently to allow the patient to walk, but it remains weak, so that it must be dragged after him. The extensors of the foot and toes, however, remain paralyzed, and, on account of the equinovarus position, the patient is forced to move the leg (the toes cannot be lifted from the floor) in a half circle around the other one. The flexors of the knee also remain paralyzed.

The arm regains its function later, but less completely. The patient generally learns to move it at the shoulder and elbow; the hand and fingers, however, remain completely paralyzed.

Contracture generally results in a hemiplegia, which is due to a direct focal lesion. We distinguish an early and a late contracture. The first may develop in the first hours or days and disappear soon after. It is probably produced by irritation of the pyramidal fibres. It has been particularly noticed when a hemorrhage breaks through towards the ventricle.

Late contractures occur some weeks after, rarely earlier than the end of the second week. It is a chronic disorder, occurring hand in hand with a descending degeneration of the motor conducting tract, but probably not produced by it. The arm is generally brought to the following position by it: The upper arm is adducted; the lower arm flexed at an acute or right angle; the hand is pronated and flexed; the fingers either flexed at all joints or only at the interphalangeal joints. A contracture with a position of extension occurs rarely. When the contracture is slight, the fingers may be extended. The leg is extended, the foot in an equinovarus position. The contracture is due to a continued muscular contraction, which disappears in sleep, and which also seems less in the morning. It can only be overcome by force, and appears again immediately. It is increased by all sensory stimulation, especially by cold. Artificial removal of the blood from the extremity, through the use of Esmarch's tourniquet, relaxes the contracted muscles (Brissaud). It is combined with *increased deep reflexes* of both arm and leg. An increase in the reflexes, which occasionally appears a few hours after the hemiplegia occurs, does not, however, indicate that the hemiplegia will follow.

Hitzig associates the contracture with the accessory movements which are described below. According to Monakow, the influence of the sensory impulses upon the lower motor apparatus, which appears markedly when the influence of the will is withdrawn, is the cause of it.

The musculature of the paralyzed side does not atrophy, and reacts normally to electricity. After some time we find a slight decrease in muscular volume, due to inactivity.

In some casés (Quincke, Eisenlohr, Borgherini, etc.), considerable atrophy was found early, and in limbs which later were movable. It could not be referred to inactivity, and some decrease of the electric excitability was present. A good explanation of this has not yet been made, none of the theories advanced being satisfactory.

The assumption that in such cases the descending degeneration of the pyramidal tract has been continued to the anterior horns (as Charcot thought) does not always hold true (Senator). This atrophy accompanying monoplegia and hemiplegia of a cerebral origin is, however, very rare. The view that there are trophic centres for the muscles in the cortex and central ganglia (Quincke) seems to me to be founded on insufficient data. Monakow has also spoken against this idea. He believes that the loss of sensory, motor, and vasomotor impulses is the cause of the muscular atrophy. *Articular* affections may exist at the same time, and the attempt has been made to refer this muscular atrophy to a secondary arthritic process (Darkschewitsch). An atrophy of neuritic origin may, of course, develop in one of the paralyzed limbs. The atrophy is, however, so slight and uncommon as not to deserve much interest.

The muscles of the non-paralyzed side often—perhaps always—suffer some impairment in strength, which may show very prominently in the leg.

Monoplegia is rarely observed in lesions of the internal capsule, though isolated facial paralysis (Diday, Duplay), also monoplegia cruralis, have been described as having occurred from such lesions; the latter is probably generally combined with sensory disturbances.

Lesions of the anterior limb of the internal capsule probably only produce paralytic symptoms when they occur near the knee of the capsule.

Diseases of the *central ganglia* produce symptoms of paralysis when they involve the internal capsule directly or indirectly through pressure. Small foci, limited to the striate (corpus striatum), or to the thalamus (thalamus opticus), may be symptomless. Diseases in the region of the thalamus were observed to produce mimic disturbances in some cases. Nothnagel and Bechterew claim that mimic movements of one side of the face are dependent upon the integrity of the thalamus of the opposite side. Small lesions may produce symptoms of irritation (involuntary laughter); larger ones, loss of mimic with intact voluntary movements.

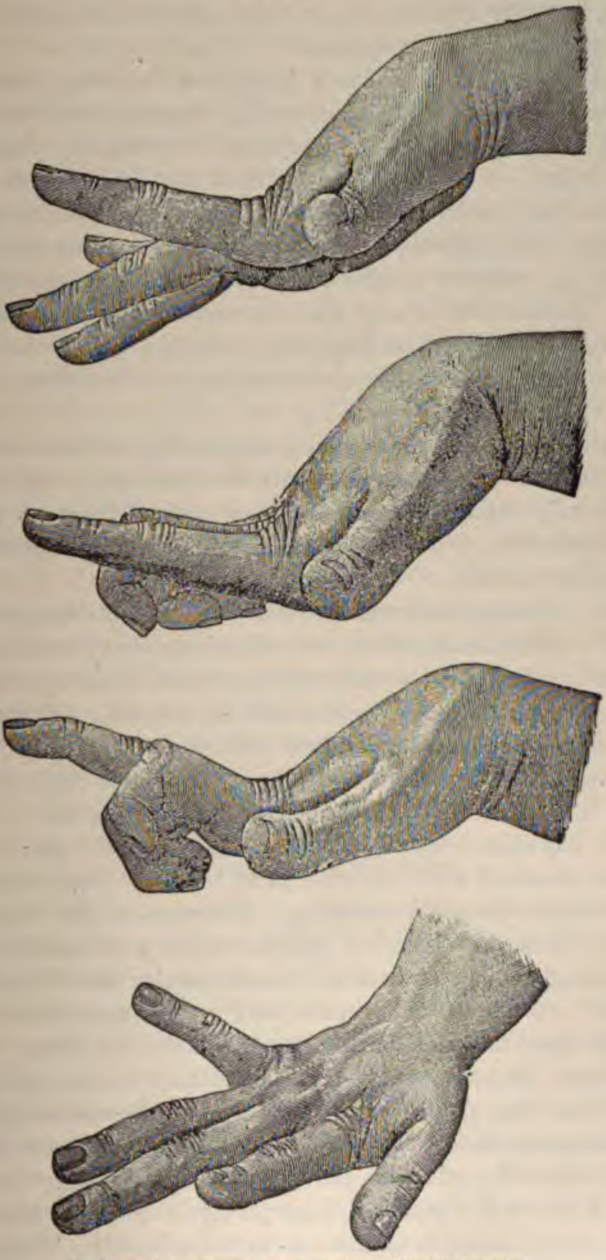
Certain phenomena of motor irritation which occur more frequently in diseases of the thalamus than in those of other regions, and which stand in close relation to hemiplegia, following and occasionally preceding it, are *hemichorea*, *hemiathetosis*, and similar disorders of motility.

Hemichorea expresses itself by involuntary movements of the limbs on one side of the body, passing from one muscular group to another, and producing a trembling and awkwardness of the limb. The form which interests us here has been called a *post-hemiplegic chorea*, because it follows a hemiplegia; a pre-hemiplegic chorea is more rare. This may develop, according to Charcot, from a slowly progressive hemorrhage in the thalamus. I observed a post-hemiplegic chorea confined to the arm in one case.

Athetosis, or hemiathetosis, is also characterized by involuntary movements, appearing mostly in the fingers and toes, while the chorea involves the whole extremity. They are slow movements of flexion and extension, adduction and abduction (Fig. 221), occurring continually, even in sleep, sometimes with interruptions, or only upon attempts at active movement or from some psychic emotion. All fingers are not moved at the same time, nor in the same manner, but in a most bizarre fashion. The fingers may be overextended and sprawling, while the hand remains in extreme flexion. Some of the fingers may be flexed, others extended, etc. These movements have been compared with those of the arms of a polyp, though they are not so slow.

Tonic contractions may also occur in the other muscles of the body. There are many grades of motor disturbances between chorea and athetosis which correspond neither to the type of hemichorea nor to that of athetosis. These disorders may also be found side by side in the same individual.

Fig. 221.



Position of the fingers in athetosis. (After Strümpell.)

The athetosis also follows hemiplegia, as a rule, especially a hemiplegia due to cerebral infantile palsy. It follows the hemiplegia in such cases, but only after months or years, and then only when a certain degree of active motion has appeared.

Hemichorea and hemiathetosis have been so often observed after thalamic lesions that many were inclined to regard them as being due to disease of this region (Gowers, Stephan, Nothnagel). On the other hand, the experience of many tends to show that it is not the lesion of the thalamus, but the irritation of the neighboring pyramidal tract which is the cause. The hypothesis has been therefore set up that these disorders may be produced by affections of the motor centres and lesions anywhere, providing it be near the motor conducting tract (Kahler and Pick). We have referred, on page 413, to another theory which connects the phenomena with the optic thalamus, and yet has them come from the cortex.

It is noteworthy that these phenomena correspond in most cases to a lesion of the thalamus lenticula (lenticular nucleus), or of the posterior limb of the internal capsule, while a cortical lesion or disease of the pons, cerebellum, and brachium conjunctivum has been observed in only a small percentage of cases.

Choreic twitchings are said to have been produced experimentally by irritation of the motor cortex with chemic poisons (creatinin).

Cases of unilateral or bilateral athetosis have also been observed, in which no alteration in the brain could be detected. These cases of *idiopathic athetosis* are often combined with idiocy.

Accessory movements (*Mitbewegungen*) have been often observed in paralyzed limbs during active movements of the muscles of the sound side, or in yawning, coughing, etc. For instance, the paralyzed hand may become clenched when the other hand is used to shake hands with, or the closed hand will open in yawning. The opposite also occurs, in that attempts to contract the paralyzed muscles will be accompanied by similar contractions upon the sound side. Senator styles these "compensatory movements." Occasionally the paralyzed upper extremity will execute movements when the patient attempts to lift the leg of the same side, or *vice versa*. These accessory movements may be very prominent in paralytic forms occurring in early childhood, even to such a degree that all movements are executed on both sides and in the same manner (Westphal).

Rarer forms of posthemiplegic motor symptoms of irritation are a unilateral *tremor* and a posthemiplegic paralysis agitans. Also a tremor similar to that of multiple sclerosis occurs occasionally. Simple, rapidly repeated twitchings in definite muscular groups, also a combined tremor and ataxia, may follow hemiplegia.

Diseases of the *cerebral peduncle* produce *typical hemiplegia*, as a rule, accompanied by paralysis of the *oculomotor* of the *opposite side*,—*i.e.*, an affection of the left crus of the cerebral peduncle produces paralysis of the left oculomotor (occasionally also partial paralysis of the right) and right-sided hemiplegia.

Lesions of the upper part of the pons produce simple hemiplegia, and only the involvement of other tracts in the pons reveals the exact seat of the lesion.

If, however, the lesion takes place just after the central facial tract has decussated,—that is, near or at the height of the nucleus, the facial will be paralyzed on the same side and the extremities on the other. This is called *alternate hemiplegia*. An alternate hemiplegia may also occur in which the hypoglossus is paralyzed on one side and the extremities upon the other, the tongue deviating towards the sound side.

Conjugate deviation of the head and eyes towards one side is a frequent phenomenon in cerebral diseases. A deviation of the head and eyes towards the unparalyzed side,—*i.e.*, towards the lesion, is frequently observed in cerebral diseases, particularly in the beginning of acute processes, accompanied by paralysis of the opposite side of the body. This phenomenon is a result of paralysis or weakness of the muscles which draw the head and eyes towards the side of the hemiplegia. In spastic conditions the head and eyes deviate towards the spastic side. In pontile diseases the eyes often deviate towards the opposite side from the lesion, due to injury to a common centre for the abducens of the same side and the internal rectus of the opposite side, lying near the abducens nucleus. The head is at the same time often turned voluntarily towards the opposite side.

DISORDERS OF SENSIBILITY.

Diseases of the motor cortical centres are often accompanied by sensory disorders in the paralyzed limbs. They rarely reach a degree of complete hemianesthesia. We generally find a parasthesia, also a slight decrease in sensation, especially on the distal parts of the extremities, which may be so slight as to demand careful searching before it is detected. Several or all qualities of sensation may be impaired—the sensation of touch particularly, and, at times, that of position. Whether the latter is always produced by an involvement of the parietal lobes, or also occurs in processes confined to the central convolutions, has not been definitely decided, though I regard the latter as probable. (Compare page 397.) In monoplegias the sensory disturbances are found in the paralyzed extremity, or a part of it. We know nothing definite concerning the occurrence of sensory anomalies in diseases of other cortical regions. Large cortical areas, even the motor zone, may be destroyed without causing a lasting anesthesia.

A peculiar sensory disorder has been observed by Wernicke, myself, and others, in lesions of the motor zone. With intact sensibility of the hand, objects cannot be recognized by touch (tactile paralysis, mental anesthesia). It appears to be a loss of those memory pictures which are acquired through the tactile sense.

I have often noticed this symptom at the first examination, to find it absent later. It was probably due to an impairment of the ability to concentrate the attention upon the paralyzed side. This phenomenon is often found in hemiplegics whose trouble dates from early childhood, and is due in these cases to the fact that tactile memory pictures were never acquired by the paralyzed hand.

Flechsig and Monakow seek to explain this disturbance by stating that the co-ordination of the elementary sensations of the skin, muscles, joints, etc., does not occur any more, and that without this co-ordination the formation of true stereognostic conceptions is impossible.

Diseases of the sensory conducting tracts entail more severe disturbances of sensation. Lesions in the posterior part of the internal capsule produce *hemianesthesia* of the other side of the body. This, if complete, extends over the entire integument and mucous membranes of one side of the body, and is limited by the middle line. I cannot agree with Grasset, who claims that the cornea is always spared. Generally, however, the anesthesia is not equally distributed, some places being more anesthetic than others. A peculiar type of hyperesthesia may be combined with the anesthesia, in that painless, even simple, tactile stimulation produces a "peculiar, disagreeable" sensation, or a lasting, intense sensation of pain with stronger stimulation.

Sensory phenomena of irritation—excluding the pain produced by the muscular contraction—occur at times in diseases of the sensory conducting path,—*i.e.*, *severe pains* in the opposite half of the body.

It appears that in such cases the alterations have not produced a complete break in the sensory conducting path.

The close relation between the sensory and *optic conducting* tracts makes it obvious that hemianesthesia is often seen associated with *hemianopsia*. Cases have also been observed in which the hemianesthesia was accompanied by impairment of the senses of *smell*, *taste*, and *hearing*, and by *amblyopia*, with concentric contraction of the visual field, on the same side of the body. Charcot therefore assumed that the sensory conducting tract contained fibres which led to the cerebral cortex from the cranial nerves, and fibres which conducted the facial impressions to a hypothetical centre for the whole retina of the eye of the opposite side.

Even though the optic and acoustic conducting tracts lie near the sensory, nevertheless, injury of the first produces hemianopsia, while a unilateral lesion of the latter does not appear to produce any lasting disturbance of function.

Whenever, therefore, the so-called mixed hemianesthesia or a hemianesthesia of the special senses is found, hysteria or a combination of an organic cerebral disease with hysteria or with some other functional neurosis is present. This is also indicated by the fact that in some cases the sensory disturbance could be transferred or removed by application of magnets (Vulpian, Bernheim, myself). Bechterew has called attention to another possibility, namely, that vasomotor tracts for the opposite side of the body are found in or near the *carrefour sensitif*, and that injury of them produces circulatory disturbances in the peripheral organs of special sense of the opposite side of the body, causing thereby an impairment of their functions. In this way a lesion of the internal capsule can produce a true "hysteric" hemianesthesia.

Diseases confined to the striate body or thalamus, without injuring the internal capsule directly or through pressure, need not produce much sensory disturbance. Affections of the crusta also do not produce any sensory disturbance. If, however, the tegmentum or the fillet be injured here, or in the geminal, pontile, or medullary region, a *hemianesthesia* of the opposite side results without involvement of the sensory organs. Only the acoustic of the opposite side (Henschen), or both sides (Siebenmann), may be affected.[1] It has not yet been determined what part of the tegmental cross-section need be disturbed to produce sensory disorder. The fillet and the reticular field come first in question (Kahler and Pick, Henschen, Moeli); the first is at least the most important part of the sensory conducting tract.

Unilateral affections of the pons and medulla, which involve the nucleus or roots of the fifth nerve, may produce crossed or alternate paralysis,—*i.e.*, anesthesia of the same side of the face and of the opposite side of the rest of the body. This hemianesthesia is at times a dissociated one,—*i e.*, it affects only the pain and temperature sense. In one of my cases, an alternate hyperesthesia appeared as the first sign of an affection of the medulla oblongata.

Diseases of the sensory centres and conducting tracts may also cause *ataxia* in the extremities of the opposite side of the body. *Hemiataxia* has been noticed in diseases of the parietal lobe, motor centres, (?) internal capsule, geminum, and pons. It has not been determined whether the same tracts, a break in which causes anesthesia, are at fault here or not. In the pons the mesial-ventral parts of the tegmentum were especially involved in those cases in which ataxia occurred (Moeli). In a case of pontile disease I found an ataxia limited to the arms.

---

[1] In one case, which I saw not long ago, a diplakusis developed after an apoplectic stroke in conjunction with hemiplegia (the patient heard the basal tone at the same time as the third).

Ataxia also occurs in lesions of the cerebellar peduncle and cerebellum.

VASOMOTOR AND TROPHIC DISTURBANCES.—They generally accompany hemiplegia and hemianesthesia, rarely occurring alone. In diseases of the motor centres which cause monoplegia, an increase and decrease of the temperature on the skin of the paralyzed extremities, especially on the hand, and also reddening, cyanosis, and even edema, are observed. Experimental observations have shown that the cortex contains vasomotor centres (near the motor ones). (See page 396.) These phenomena have been observed even oftener in diseases of the conducting tracts than in ordinary capsular hemiplegia or hemianesthesia. It is probable that the vasomotor conducting tract lies near the sensory. The edema is, as a rule, confined to the paralyzed limbs, and may even affect this side exclusively in cases where a nephritis exists at the same time. In many cases a tendency to decubitus exists, especially in cases of severe hemiplegia. Other trophic disturbances may occur, as eruptions of pemphigus vesicles, unilateral graying of the hair, etc. Gangrene of the paralyzed limbs was observed once (Préobragenski).

The *joints* of the paralyzed limbs become affected in many cases. The inactivity and fixation of the limb in one position causes the articular disorder, but it can develop in another way: from one to four weeks after the hemiplegia has come on, or later, when some movement has returned, an acute or subacute arthritis develops, with reddening and slight swelling of the joints. Hemorrhages occur rarely with it. It can be assumed that trophic influences are at play in such cases. We have already referred to the relative frequency of articular disorders with muscular atrophy in hemiplegics.

VISUAL DISTURBANCES.

It is hardly possible to distinguish such ocular disorders as are produced by disease of the optic nerve from such as are of cerebral origin, and, moreover, as the optic nerves are genetically a part of the brain, we are justified in treating of the diseases of these nerves at this place. The importance of ophthalmoscopic examinations need scarcely be dilated upon. Not to employ them in cerebral diagnosis would mean to close up the only window through which we may peer into a veritable chamber of mysteries.

Excluding the developmental anomalies, alterations which may be detected ophthalmoscopically are *optic neuritis*, or *choked disk*, and *atrophy*, which manifests itself in various forms.

We speak of choked disk when a noticeable steep prominence of at least two-thirds of a millimetre (refractive difference, two diopters) is present. It is characterized by cloudiness and swelling of the papilla, as well as by indistinct borders to the same. If marked, the papilla is very prominent, reddish or grayish-red, cloudy, and without defined borders; the arteries are contracted, the veins dilated, and the vessels invisible at some places. The diameter of a papilla may be increased threefold.

Hemorrhage and white spots (fatty degeneration) also occur. (Fig. 222.) The distinction between optic neuritis and choked disk is, however, *not always* easily made. Some authors are inclined to entirely separate the inflammatory alterations from choked disk.

Choked disk may be confused with an *albuminuric retinitis* when the latter is confined to the papilla. Also, congenital anomalies occur which are very similar to an optic neuritis,—*i.e.*, a congenital optic pseudo-neuritis (Wecker, Bristow, Uhthoff).

Sight may be normal even though a choked disk is present; in advanced cases, however, it becomes involved, an irregular concentration of the visual field and a decreased central sharpness of vision manifesting themselves. These may increase to complete blindness; an amaurosis suddenly appearing, to disappear as quickly, and later to reappear repeatedly, has been observed in choked disk. It is probably a result of a periodic increase in intracranial pressure (tumor cerebri, etc.), which corresponds to a compression of the optic chiasm through a flow of fluid into the third ventricle.

Optic neuritis occurs in *chlorosis*, after the *acute infectious fevers*, and in *lead poisoning*, in the latter it is true more as a symptom of a general cerebral trouble (saturnine encephalopathia).

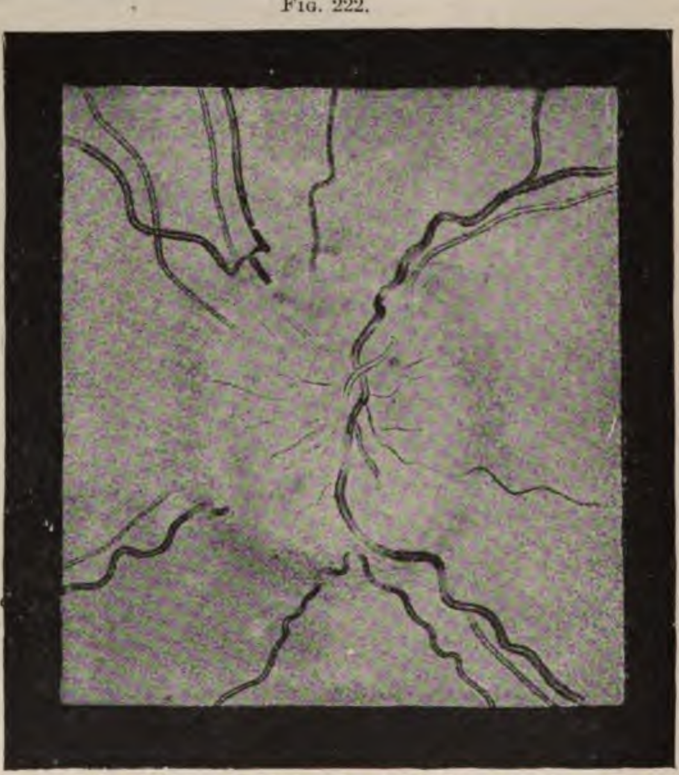
The most important and frequent cause of optic neuritis, or choked disk, is a brain tumor. In such cases it is generally present in both eyes, and its development is independent of the location of the neoplasm.

Internal hydrocephalus often produces choked disk. It is also not an uncommon symptom in *cerebral abscess* and sinus thrombosis. An optic neuritis occurs more rarely in the acute non-purulent *encephalitis*. It is a frequent symptom of brain syphilis and of the different forms of (basal) *meningitis*. I observed a choked disk once in a chronic recurrent hemorrhage, though it has often been observed in *hemorrhagic pachymeningitis*. A rheumatic optic neuritis is also said to occur.

It has rarely been observed in ulcerative endocarditis (Mackenzie, Broadbent). It occasionally occurs in cranial anomalies, for example, accompanying the so-called steeple-shaped cranium or oxycephalus.

The nature of choked disk—the name is due to Graefe—is not yet clearly understood. According to some (Schmidt-Rimpler, Manz), it is due to the cerbrospinal fluid under increased pressure forcing its way into the optic sheath, and so compressing the veins as to produce a true congestion or edema, which may be accompanied by inflammation. According to others (Leber, Deutschmann), toxic products are the direct cause of the inflammation of the papilla. Other theories, as those of Adamkiewicz, Parinaud, Jackson, etc., need not be discussed here.

FIG. 222.



Optic neuritis from a brain tumor (or papillitis). (After Gowers.)

The choked disk may disappear completely,—*e.g.*, in cerebral syphilis or successfully treated brain tumors. The opening of the cranium, even in inoperable cerebral tumors may cause a disappearance of the choked disk.

If the choked disk continues any length of time, atrophy occurs.

The *optic atrophy* may be *secondary*,—*i.e.*, be preceded by an optic neuritis, or produced by a compression of the optic nerves at a point some distance behind the eyeball,—or it may be a *primary* condition.

Primary optic atrophy, which is almost always *bilateral*, occurs especially in tabes and in paralytic dementia, perhaps also rarely as a direct symptom of syphilis, and (apparently), in some cases, as an independent affection. The atrophy may be recognized by the marked pallor, the white discoloration or decoloration of the papilla, on account of which the border is more sharply defined than normally. It should, however, be remembered, that the color and vascular supply of the papilla vary greatly in health, and that the central part is, as a rule, paler than the peripheral. An ophthalmoscopic diagnosis of incipient atrophy should be made with caution, and repeated and exact observations are demanded.

Secondary atrophy, produced by compression or injury of the optic nerve (tumors, fractures, gunshot wounds, carious processes, etc.), causes a unilateral affection which may increase to blindness; in these descending atrophies the visual disturbance precedes the ophthalmoscopic alteration.

Optic atrophy in *multiple sclerosis* is almost always a partial one and rarely becomes complete. The visual disturbance, also, almost never becomes a lasting and complete one. Though it may be of neuritic origin, a marked optic neuritis is rarely observed in multiple sclerosis.

Retrobulbar neuritis is generally—though not invariably—of toxic origin, and produces symptoms of toxic amblyopia. This is the form which frequently occurs in polyneuritis. It may also come on after the acute infectious diseases.

The chief cause of toxic amblyopia is chronic alcoholic poisoning, next nicotine poisoning; quinine, carbon disulphide, etc., rarely come into question. Salicylic preparations, carbolic acid, and other poisons have been regarded as causes in some cases. The characteristic visual disorder is a bilateral, relatively central, scotoma for red and green in one zone, which extends outward from the fixation-point and involves this as well as the blind spot. At times we find a smaller one for blue, and exceptionally an absolute one for white. The central visual acuity is more or less decreased. This visual disorder is due to an interstitial neuritis of the *papillomacular* fasciculus in the optic trunk (Samelson, Uhthoff, and others). The ophthalmoscopic examination may be negative, but generally a partial atrophy of the optic nerves—a paling of the temporal half of the papilla—is found. A distinct optic neuritis is rare.

Retrobulbar neuritis of non-toxic origin has an obscure etiology. We find an hereditary form, which attacks several members of the same family, and occurs, as a rule, in youth (between the ages of twelve and twenty years). An absolute central scotoma is the condition generally found; the disorder may also confine itself to one side. It may commence with orbital pain.

A transitory amblyopia, probably depending upon retrobulbar neuritis, has been observed several times during the period of lactation. An hereditary form of optic atrophy has been described by Leber. There are also certain hereditary, familial nervous diseases, which are characterized by a combination of idiocy with amaurosis and optic atrophy (Sachs and others).

Lesions of the *optic chiasm* and of the *optic tract* need not produce any alteration of the background of the eyeball, though a descending atrophy may occur if they persist any length of time. In lesions of the chiasm, the middle piece, which contains decussating fibres, is particularly often involved. The corresponding loss of function is a *temporal hemianopsia* produced by *amblyopia* of the *mesial halves of both retinæ*. By extension of the process, total blindness or unilateral blindness with hemianopsia of the temporal side of the other eye may occur. Only rarely, however, do we find a nasal hemianopsia.

The diseases which involve the chiasm are generally neoplasms, especially syphilitic ones, also tubercular and other tumors of the hypophysis, etc. Hydrocephalus of the third ventricle may, from protrusion of the floor, involve the middle piece of the chiasm and cause bitemporal hemianopsia. It has often been observed in acromegalia.

Diseases of the optic tract cause *homonymous bilateral hemianopsia*. Fig. 201 will show how it arises. Diseases of the whole intracerebral optical conducting tract and of the visual centre also produce this condition. This symptom has, therefore, on the whole, only a slight localizing diagnostic value; the accompanying symptoms, however, render it possible to localize the process. Lesions of the tract are, as a rule, accompanied by disturbances of other basal cranial nerves, as it is rare for an affection to limit itself to the tract. Diseases of the thalamus or of the external geniculate bodies, which express themselves by hemianopsia, involve generally the internal capsule and produce hemiplegia, hemianesthesia, etc., on the same side as the hemianopsia.

Diseases of the cortical visual centre may produce isolated hemianopsia. We will indicate later some peculiarities of this cortical form. In the right hemisphere, a lesion of the optic radiations produces, on the whole, the same phenomena as one of the visual centre. In the left, the optic conducting tract follows the same course through the temporal, parietal, and occipital lobes, but passes regions which are intimately associated with speech, so that a break in it produces not only right hemianopsia, but also aphasia. The patient is generally conscious of his hemianopsia, particularly so if he is intelligent. He tries to guard against running into things by turning his head towards the blind side. Right-sided hemianopsia renders reading difficult, but not impossible.

Hemianopsia is at times combined with concentric contraction of the visual field, which generally affects the visual field of the eye of the other side. It should be remembered, however, that the temporal, that cal disturbance of the cortex of the cuneus and of the calcarine fissure), in the optic radiations, perhaps also extensive injuries of the gemina or geniculate bodies, will produce total blindness.

In this amaurosis, the pupillary light reflex is not impaired when the break in the continuity occurs beyond the reflex centre. The view of Henschen, that the macula remains free when the lesion spares the calcarine fissure, has not remained undisputed.

Küstermann calls attention to the fact that in these cases of bilateral hemianopsia paralysis of the ocular muscles, which according to our present ideas should occur, is absent.

Harris claims that a unilateral affection of the cuneus of sudden onset may produce a transitory total amaurosis, due to inhibitory influence of the lesion upon the centre of the sound side. This amaurosis, then, rapidly passes into an homonymous hemianopsia.

It can be assumed that certain forms of transitory blindness are produced by a *functional* or *toxic* paralysis of the optic cortical areas. The transient *uremic amaurosis*, in which the pupillary light reflexes remain normal, belongs here. Some authors, however, refer it to a disease of the optic nerve (edema). Perhaps there are a central and a peripheral form. A transitory amaurosis is also observed in *lead poisoning*. *Quinine* amaurosis may also, perhaps, be included here. Atrophic alterations of the papilla have, however, been noticed in long continuation of the latter. The meaning of the visual disorders occurring after severe loss of blood is undetermined. In some cases it produced symptoms of a retrobulbar neuritis.

Hemianopsia of temporary duration may also be produced by functional disorders, or by those capable of rapid retrogression. It may occur in migraine, as an accessory symptom of scintillating scotoma, also as a temporary symptom of uremia and lead poisoning. It may also form a symptom of paralytic attack.

The form occurring in migarine is probably due to vascular spasm.

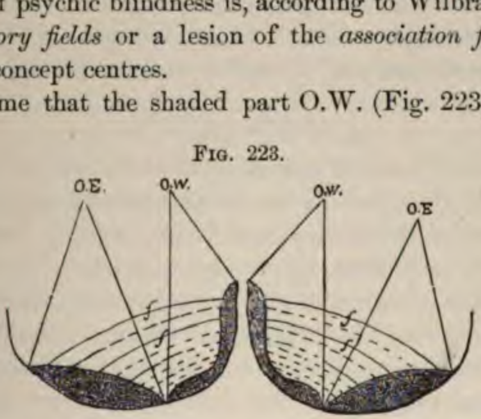
Excluding these forms, it is generally a chronic condition, caused by hemorrhage, softening, inflammation, neoplasm, etc. A transitory hemianopsia is said to have been observed immediately after an apoplectic attack as an indirect focal symptom. In one of my cases the hemianopsia has existed for twenty-seven years without other symptoms. In another case it was of congenital origin.

MENTAL OR PSYCHIC BLINDNESS.—This peculiar visual disorder, first observed by Munk in animals, has also been noticed in man. In this condition objects can be seen, but their use cannot be comprehended. The patient sees with his eyes, but no longer with his brain, as a patient of Wilbrand expressed himself. A knife, key, etc., are seen, but their use is not recognized. Previously well-known streets and rooms are still seen, but appear strange. The visual memory pictures are at the same time affected,—*i.e.*, he can no longer spontaneously reproduce facial impressions, or imagine the appearance of objects, landscapes, physiognomies, etc., previously known to him. The memory pictures are, however, in some cases intact. Psychic blindness should not be confused with visual aphasia (see next chapter), with which it may, however, be combined. A person afflicted with psychic blindness is generally unable to measure distances. In most cases psychic blindness was combined with homonymous *bilateral hemianopsia* of one side, and, at times, partially on the other.

The cause of psychic blindness is, according to Wilbrand, a lesion of the *visual memory fields* or a lesion of the *association fibres* connected with the optic concept centres.

Let us assume that the shaded part O.W. (Fig. 223) represents the visual centre in both occipital lobes; there is another cortical region—probably the convex surface of the occipital lobes, perhaps also parts of the lower parietal lobe—in which the memory pictures for objects seen in life are stored. We will call, with Wilbrand, the region O.E. the *visual memory field*; what is received in O.W. is only comprehended after exciting in O.E. the corresponding memory pictures. If, therefore, O.E. is destroyed or injured, or if the conducting paths between O.W. and O.E. are broken, the patient sees without recognizing. If O.W. on both sides is impaired, the patient is blind, cortically blind. If O.W. is injured on one side, and on the other side O.E. or *f*, we would find unilateral hemianopsia and psychic blindness.

FIG. 223.



This view of psychic blindness is opposed to that of Lissauer. According to him, the cortical field of the occipital lobe is to a certain extent only the retinal half of the cortex. The conception of space, the recognition of objects, only occurs when the retinal field of the cortex is associated with other mental centres, especially with those for ocular movements; only the combined action of these different centres produces any recognition. He distinguishes two forms of psychic blindness: (1) an apperceptive, in which the occurrence of conceptions appertaining to the senses are involved; (2) an associative (transcortical), in which, from disturbances in the association tracts, the connection between the retinal fields of the occipital lobes with the centre of one or more other senses is interrupted.

Bilateral diseases of the occipital brain are therefore generally the cause of psychic blindness. In twenty cases which came to a post-mortem, this was found in twelve of them. Only in a few cases has psychic blindness been observed in unilateral disease of the occipital lobe. In these cases it was a neoplasm, which acted upon the occipital lobe of the other side also.

Psychic blindness may disappear when caused by reparable alterations (traumatic encephalitis, gumma, etc.). It is a transitory symptom in paralytic dementia. It may probably also be due to functional inhibition. It is generally incurable. It may be simulated by a condition of visual weakness accompanied by achromatopsia (König and Siemerling).

SPEECH DEFECTS.

The two chief forms of speech disturbance, *anarthria* and *aphasia*, have been briefly described in the general part of this work. The first is a disturbance of articulation, and is produced by bilateral paralysis or paresis of the articulation musculature,—*i.e.*, of the muscles of the lips, tongue, palate, larynx, and of the respiratory muscles. Unilateral affections of the facial and hypoglossal centres, and of the conducting tracts passing from them, involve the articulation but slightly, and the disturbance soon disappears. One hemisphere seems, however, in some individuals to have more influence than the other in the innervation of the muscles of speech.

The typical speech defect in cerebral diseases is aphasia,—*i.e.*, the loss or impairment of the ability to express ideas with words or signs, and to comprehend spoken and written language so that the corresponding ideas will be awakened. This is the definition of aphasia in the widest sense of the word. It may be subdivided into various components, of which some have been given special names. *Alexia* is understood to be the loss of the power to read with intact sight; *agraphia*, the loss of the power to write without impairment of the movements of the arms. The most important factor—and, as introduced by Trousseau, aphasia only meant that—is the loss of speech. Let us analyze this further. A person previously healthy has a cerebral hemorrhage. On the return of consciousness he can no longer express himself by words. Although the lips, tongue, etc., may be moved, or are only paralyzed upon the right side, although sounds may be formed, although, as we can see from the gesticulations and the facial expression, concepts are still formed in the brain, the ability to convert them into words is lost or impaired. Generally a few words, as *yes* and *no*, or a senseless collection of syllables, as *bibi*, *lala*, *tatata*, etc., are at the patient's service; but these represent all of spoken speech that is retained. Sometimes the last word, or a short sentence that was spoken just before the paralysis came on, forms the only remnant of speech, and is repeated in a stereotyped manner. *Anarthria* is due to paralysis of all the articulating muscles, and dysarthria is but a slighter degree of this disturbance. But in aphasia the central part of word-formation, the conversion of concepts into words, is lost or impaired. If a question is asked of such a person, he makes a gesture expressing his difficulty, points to his mouth, and gives one to understand that he is not able to speak. Or he pronounces constantly the few words or syllables of which he is capable, as with one of my patients who gave expression to all his thoughts and knowledge with the words "thanks" and "nose." Generally such persons seek to make themselves understood through gestures, but often the ability to express themselves through mimic movements is also annulled (amimia).[1]

There are cases in which the defect only refers to the production of words; this form was later called *motor* or *ataxic* aphasia; ataxic, because in it attempts are often made to produce words, but false letters and syllables are introduced, others doubled, transposed, or misplaced. It is, however, better to allow this name to fall into oblivion.

Broca, in 1861, showed that motor aphasia was produced by a lesion of the posterior part of the third left frontal convolution. Complete motor aphasia is almost always accompanied by paralysis of the right side of the body, though I observed one case in which, notwithstanding absolute mutism, no paralysis was present.

In other cases, another disturbance is found accompanying the motor aphasia. The patient hears without understanding what is said, it is as if he were hearing a foreign language which he does not know, or of which he only comprehends a few words. We can ask him his name, age, or occupation, to shake hands, etc., but he does not respond. He acts like a foreigner who does not understand our language, or understands some and errs in the rest. Naturally, this confuses and depresses the patient and impairs his range of thought.

This *defect* of *perception*, which also occurs isolated, is called *sensory aphasia* (Wernicke), or *word-deafness* (Kussmaul). When it exists alone, the patient can speak, can express his ideas, without understanding what is spoken by others. Closer observation reveals, however, that the power of producing words is also impaired. Sensory aphasia depends upon the loss of the memory pictures for the sounds of the words. Every word entering our ears leaves such a sound picture in the brain. We are able to excite this voluntarily, to sound this word within ourselves, and in order to use this sound picture we think of a word. In mechanical speech, most persons do not find it necessary to revive this word-sound—it is otherwise in thinking or seeking after a word. Thus it occurs that sensory aphasia, the loss of the sounds of words, produces more or less impairment of speech. The patient uses a series of words or whole sentences without any difficulty. As soon, however, as he finds it necessary to seek after a word, his inability to sound words within himself becomes evident; he feels around, producing false, similar-sounding words, or words similar in sense. Spontaneous speech is therefore easier for him than a conversation. A question or demand will reveal his defect, because the word coming from without disturbs his centre for sound pictures, and as it does not functionate properly, spontaneous speech becomes impaired.

---

[1] A deaf and dumb patient, who was able to make himself understood by the use of his fingers (sign language), lost this ability, after disease of the left hemisphere, for the right hand, although this was not paralyzed (Grasset).

A patient of mine with sensory aphasia will say, "Doctor, I did not sleep well last night, I do not feel well, I hope to be soon well." After awhile, I ask, "Mrs. A., how are you?" "Oh, doctor, I have different feelings . . . wose, so wors . . . not worse . . . I am again better worse." She understands the sense of my question, for which she is prepared, and which is often asked her. But the diseased centre for sounds is irritated, produces false sounds, which influence her spontaneous speech.

In many cases those suffering from sensory aphasia can only produce an incongruous jumble of words, like a new language. An impulse to speak may accompany this. It is possible that a lesion of the centre for sound is present, which not only paralyzes but also irritates, and in this way serves to excite the production of senseless words.

The insertion of wrong words, the interchange of words, is called *paraphasia*, and this is generally combined with sensory aphasia.

The impairment of the ability to awaken tone pictures in oneself, to remember words, whenever it occurs isolated and is not combined with a disturbance of word-perception, has also been called *amnestic* aphasia. It depends upon the fact that the sensory speech centre cannot be called into action by other higher psychic centres, though, when stimulated through the acoustic, it may still functionate.

Some have discarded the term amnestic aphasia, or confined it to those cases in which a general disturbance of the memory was the cause of the verbal amnesia (Lichtheim). I would desire, however, with Eisenlohr, to retain the above conception of amnestic aphasia.

The localization of the centre for sound or tone pictures in the first convolution of the left temporal lobe is due to Wernicke's able investigations. He showed that the diseases of this region produced sensory aphasia.

Motor aphasia is often combined with *agraphia*, often even when the right hand is not paralyzed. It has been attempted to explain this by assuming that writing is preceded by an "internal speech," and that therefore the inability to produce words must also be shown by the want of a graphic speech. Agraphia is, however, by no means a necessary concomitant of motor aphasia, and it is certain that, in many persons, motor agraphia is independent of motor aphasia.

I treated a painter who has been completely aphasic for seventeen years as a result of an apoplectic stroke, and could only say yes and no, and who, nevertheless, was, during the entire course of his illness, able to describe it in writing, and who painted very nicely.

Bastian combats the idea that a lesion of Broca's centre produces agraphia. The view that the loss of the idea of the movements used in writing is the cause of this agraphia is not satisfactory, as those affected cannot form words from letters placed before them (Mirallié).

Agraphia is more often combined with sensory aphasia. It is then a verbal one. This is explained by assuming that, in many persons, the sound of a word must appear before its appearance in writing can be grasped, and that the impulse for movements necessary to the production of the letters of the alphabet follows this. A special centre for graphic movements has been located in the second left frontal convolution (Exner, Charcot, Pitres), but probably does not exist. It is more probable that the transference of such impulses occurs in the motor centres, most often in the motor centre for the right hand. This must be directly connected with the visual sphere by a tract, as one is able to copy what he does not understand or comprehend.

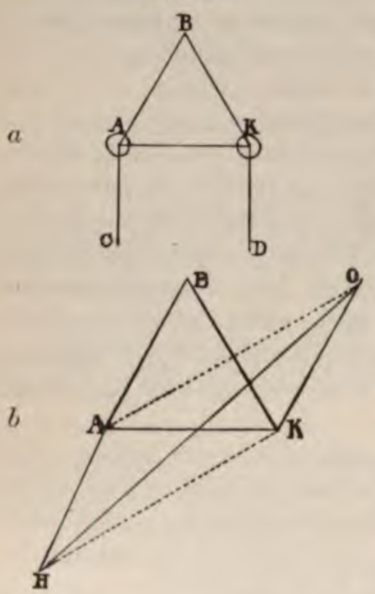
Alexia (word blindness) generally accompanies sensory aphasia. The memory pictures for graphic signs have been assigned to both visual spheres. In order, however, to make them available for reading, most persons must produce the corresponding verbal tones. For example, in putting together the letters l, o, v, e, to form the word *love*, it sounds within us, and is only then perceived. An injury to the centre for the sound pictures must, therefore, injure the ability for reading. Many persons, especially the uneducated, read aloud; in these, a lesion of the motor speech centre may also produce alexia.

Alexia is also an isolated symptom in some cases. As a cause of this, a lesion of the left lower parietal lobe (gyrus angularis) has generally been found, and it has therefore been assumed that the conducting paths from both visual spheres pass through the left lower parietal lobe to reach the centre for the tone pictures. According to Dejerine, the left lower parietal lobe contains a special centre for the pictures of the alphabet (this is very improbable). Bastian distinguishes between a parietal and an occipital alexia.

These disorders are better understood by analyzing the mechanism of speech by means of a schematic representation. We must, however, remember that all diagrams are somewhat artificial, and that all forms of disease cannot be put into them. We are indebted to Wernicke, Lichtheim, Charcot, and others for schematic representations of speech. Though they do not entirely harmonize, they have shown us the way of acquainting ourselves with the mechanism of speech. Only the most important facts will be given.

K, in Fig. 224*a*, represents the centre for the sound of words. The words enter the ear, passing along the acoustic tract from D, and leave behind memory pictures in K. The workshop for the production of words is at A. The sounds of words stored up at K are imitated until a similar sounding word results. This occurs in A under control of K, until finally the concepts of verbal movements in A become so independent that they can be directly produced from the concept centre B. Many observations seem to indicate that the tract BKA is used in spontaneous speech also. The conducting path connecting A and K appears to pass through the island of Reil. From A, the impulses pass to the nuclei in the medulla oblongata, perhaps first to the cortical centres for the muscles of articulation at the foot of the central convolutions, which govern the movements of the muscles of speech. For the sake of simplicity, the concept centre is regarded as being single, although this is not true. The formation of concepts is a complex process, and is connected with the whole cerebral cortex.

FIG. 224.



Verbal concepts, for instance, contain *motor*, *visual*, and *auditory* memory pictures. The word *bread* is composed of a graphic picture, a sound picture, a motor memory picture for the muscular movements needed to speak the word, and another one for the movements of the hand necessary to write it.

The concept *bread* is composed of a visual picture (we see the bread before us), of a memory picture of the taste of bread, and of tactile sensation (in touching and chewing the bread), etc.; also of the word-memory pictures indicated above.

This shows that the concept of a concrete object is not connected with one centre, but that it is fed by different mental spheres, by a summation of memory pictures of perceptions belonging to the senses, and that it can be directly or indirectly excited through these.

A concept may be awakened by an allied one. A view of a wheat-field awakens the concept *bread*, etc. The recognition of the fact that the memory consists of many partial memory centres teaches us that a speech defect may occur from an interruption in the tracts which connect the sense centres with the speech centres, and is therefore of the utmost importance. Even the simple figure 224*a* shows that many modifications of motor and sensory aphasia can occur, according as the disease is localized in A, K, or in the tracts, AK, AB, KB, AC, KD. To illustrate, a lesion of A produces motor aphasia, and at the same time makes repetition of speech impossible, while a division of AB destroys the power of spontaneous speech, but does not impair repetition of speech.

The consideration of graphic speech makes our diagram more complete. Tracts must pass from the visual sphere—which is connected with the outer world by the optic nerve, which for simplicity is represented as a centre, O, connecting it with other centres. (Fig. 224*b*.)

It probably is always connected with K, and this connection renders reading possible, which occurs by way of OKB, or, if reading aloud is indulged in, by way of OKBA.

A direct tract, perhaps, passes from O to A in some individuals. They convert visual pictures directly into motor ones, and only understand what they read when they read it aloud; the process, therefore. passes along OAB, perhaps OAKB. Even a direct connection between O and B may exist. Graphic symbols, as figures, are not first translated into sounds, but are directly transmitted from the graphic picture to the concept. A direct tract must pass from O towards the motor centre for the right hand (OH).

Spontaneous writing, according to some authors, presupposes a combination between A and H. Probably the tract BKOH is generally in question, as, in addition to the tone picture, the visual memory picture of the written letters must first appear before it can be transmitted to the motor centre.

The agraphia is absolute—the patient is not able to draw a single letter—or partial. Some are still able to write their names, others are capable of producing a few words or letters in writing. If the ability to write spontaneously or upon dictation is lost, the capacity for copying may still be retained. Paragraphia may also occur.

According to present conceptions of such connections, the different forms of aphasia have been grouped differently, and paradigms have been constructed which show how almost all of the theoretically constructed forms of speech defect occur.

Lichtheim divided them into *nuclear* and *conducting* aphasias, and Wernicke into *cortical*, *subcortical*, and *transcortical* motor or sensory aphasias.

In this manner the following subdivisions may be made:

1. CORTICAL MOTOR APHASIA. (Seat of the lesion in A.) Broca's aphasia.

Voluntary speech	lost.
Repetition of speech	
Reading aloud	
Voluntary writing	
Writing from dictation	
Copying	present.
Comprehension of speech	
Comprehension of writing	

The latter is impaired, according to Dejerine.

2. SUBCORTICAL MOTOR APHASIA. (Break in A C.)

Voluntary speech	lost.
Repetition of speech	
Reading aloud	
Reading	present.
Writing	
Speech comprehension	

Here the motor memory for speech is intact, but the construction of the words from letters and syllables is impaired, so that a kind of literal paraphasia occurs. This condition, to a certain extent, forms a bridge between aphasia and dysarthria.

Pitres believes that lesions in the medullary substance below the speech centre produce the same form of aphasia as the cortical, while one deep within the brain substance, especially in the left internal capsule, produces this kind of speech disturbance.

3. TRANSCORTICAL MOTOR APHASIA. (Break in A B.)

Voluntary speech	lost.
Voluntary writing	
Repetition of speech	present.
Reading aloud	
Copying	
Writing from dictation	
Comprehension of speech and writing	

4. CORTICAL SENSORY APHASIA. (Seat in K.)

Voluntary speech intact, but verbal aphasia.
Copying, present.

Repetition of speech	lost.
Reading aloud	
Voluntary writing	
Writing from dictation	
Comprehension of speech	
Comprehension of writing	

5. SUBCORTICAL SENSORY APHASIA. (Pure sensory aphasia, according to Dejerine.)

Voluntary speech	present.
Voluntary writing	
Copying	
Comprehension of writing	
Comprehension of speech	lost.
Repetition of speech	
Writing from dictation	

Freund has called attention to the fact that a bilateral labyrinthine disease may produce a difficulty in hearing words, though noises can still be heard. This condition may simulate a word deafness, and particularly give rise to confusion with the subcortical sensory form.

6. TRANSCORTICAL SENSORY APHASIA. (Break in BK.)

Comprehension of speech	lost or impaired.
Comprehension of writing	

Voluntary speech present, but paraphasia.

Repetition of speech	present, without comprehension.
Reading aloud	
Copying	
Writing from dictation	

Voluntary writing present, but paragraphia.

7. CONDUCTION APHASIA. (Break in AK.)

Comprehension of speech	present.
Comprehension of writing	
Voluntary speech	present, but paraphasia.
Reading aloud	
Repetition of speech	
Voluntary writing	present, but paragraphia.
Writing from dictation	

Copying, present.

Monakow is opposed to the idea of conduction aphasia, and calls attention to the fact that a purely cortical disease, in an anatomic sense, is rare, and that the anatomic meaning of the so-called transcortical aphasia is still uncertain.

In constructing these paradigms originally, the question of *individual* variations was not thought of. One person uses his visual memory pictures in speech more than another;. another, his auditory,—speaking, reading, and writing through the sound images; and a third, the motor. According as the one or the other of these rules, does the effect of the disease vary; the loss of a certain centre will cause a hardly noticeable disorder in one and a severe and persistent disturbance in another. Further, a complete break and loss is not always the case, a partial lesion of a region occurring very often. Just such cases cannot be grouped in the above paradigms. In such conditions, spontaneous stimulation of the speech centre may be imparied, but excitation from a sense sphere may call it into action. The patient may not be able to find spontaneously the word *watch*, but the sight of one will cause him to pronounce it, or he will repeat the word after it has been spoken to him.

Emotional excitement is also a stimulator of the speech apparatus, and may excite it to the production of words and syllables when spontaneous speech has been thought lost. Some patients who generally do not utter a word will swear when angry. Another phenomenon to be noted here is the fact that aphasics may sing at times, and, in singing, find words, but they cannot speak.

I have noticed that aphasics who are unable to comprehend written or printed matter or are only able to read mechanically combine the right melody immediately with a song text (or notes) placed before them. The localization of this disorder (Edinger) has not yet been accomplished. The observations which are said to show the presence of a "sensory amnesia" without a corresponding speech disturbance are not conclusive to me.

Aphasia may be due to the inability to retain a memory picture long enough to allow it to be transmitted to the speech centre. For instance, a patient of Grashey's could not read; when the letters of a word were seen singly and successively, he had forgotten the first one when he came to the second. Strümpell describes a form of aphasia due to associative disorder.

Another factor to be taken note of is the confusion caused by the appearance of false pictures of sounds, print, and words. This is especially shown where the patient is required to find the words for different objects in succession. After he has found the word "food," "suit" will be styled "sood." The word "cart" is heard, and immediately the word "acarat," etc., is pronounced.

A recognition of the defects of a diagrammatic representation of this subject has induced some authors to formulate a new theory as to the nature of speech defects, in which the conception of speech centres in the old sense of the word is departed from, and in which the different association tracts between the cortical fields of the sense and motor centres pass a ganglionic point, so that the lesions occurring here divide several such tracts.

The old idea of speech centres should, however, be retained. We must remember that they are connected in various ways with each other and the central centres, so that injury of one impairs, more or less, the functions of the others, and that individual moments play a prominent part in the acts of speech.

We can distinguish the following types.

1. MOTOR APHASIA,—*i.e.*, more or less complete loss of speech, with intact or only slightly impaired speech comprehension. As a rule, the mutism is not absolute, a few words or syllables being retained. Writing, reading, and repetition are lost or present, according as the central acts governing writing and reading are transmitted or not. Sentence formation is always impaired. The seat of the disease is, in right-handed persons, in the posterior part of the *third left frontal convolution* or in the white matter just below this. In left-handed persons, the speech centres are probably in the corresponding parts of the right hemisphere.

Bastian has described a case in which a left-handed person was affected with aphasia and hemiplegia dextra following upon a lesion of the left hemisphere.

2. SENSORY APHASIA,—*i.e.*, complete or partial loss of the power to understand spoken language with intact hearing. Spontaneous speech is generally possible, especially the mechanical form; but more or less impairment of speech in seeking after words is present; finally there is considerable disturbance when the individual is accustomed to speak only with the help of tone pictures. At times we find a marked impulse for speech with production of new and senseless words. Very often alexia and occasionally agraphia are present. The ability to repeat words is present or lost. Differences are in part produced by the intensity, place, and extension of the disease, especially, however, by individual variation. The seat of the disease in right-handed persons is in the *first convolution* of the *left temporal lobe.*

3. ISOLATED ALEXIA or WORD BLINDNESS,—inability to read, with normal speech and comprehension. Writing is impossible or possible (Dejerine). In the latter case (subcortical or pure alexia), he may frequently read writing, tracing the letters with his fingers and thereby bringing these sensations of movement to his consciousness. Cases of pure alexia present on autopsy lesions of the posterior occipital or the *left lower parietal lobe.* Probably the real cause is a lesion of the association tracts between the visual sphere and the sensory speech centre (particularly the inferior longitudinal fasciculus).

Very often hemianopsia dextra is present.

4. ISOLATED AGRAPHIA is rare. A special centre to control the memory of the movements necessary to the production of writing-symbols does not appear to exist. In persons who write by help of the visual memory pictures, a division of the conducting tracts which pass from these to the left motor centre appears to produce isolated agraphia. I saw it occur once unaccompanied by any other symptoms in a case of tumor of the left upper parietal lobe. If we assume that an inner speech precedes writing, a division of the path which passes from the motor speech centre to the motor centre of the right hand could produce isolated agraphia. Then, however, writing could eventually be possible with the left hand or with other parts. Monakow believes that every extensive disturbance in the inner formation of words produces agraphia.

5. VISUAL APHASIA (Freund) is rare. In it objects may be seen and recognized, but cannot be called by name, although the patient can speak and can find the proper word if the stimulation can be excited through the other senses. Example: He cannot think of its name

if a watch is held before him; he knows, however, that it is a watch. He finds the word if the watch is held to his ear. The patient is unable to bring the visual memory pictures into use for speech. It appears to be caused by lesions at the border of the left occipital and temporal lobes, and so extensive as to divide the conducting tracts leading from both posterior lobes to the centre for the tone pictures. The disorder is therefore mostly (or always) combined with alexia, hemianopsia, and often with sensory aphasia.

I have observed many cases of this kind and found that optic or visual aphasia is a not uncommon symptom of otitic cerebral abscess. In one of my patients, whose visual aphasia was caused by a softening in the occipitotemporal part of the left hemisphere, the aphasia persisted after the original trouble had disappeared, in that the patient was compelled to look at an object for some time before he found the proper word for it.

We can also speak of a *tactile aphasia*, though it is very rare. Although sensation is intact, objects cannot be recognized by touch, because the sensations of the tactile sense cannot be transmitted to the speech centre.

Pure forms of aphasia are rare. Most cases are of a mixed type, a loss of speech, and of the comprehension of speech occurring at the same time. The loss is generally an incomplete one. If the motor character of the speech disturbance is in the ascendancy, the patient has only a few words or letters still at his command; if the sensory moment is ascendant, he speaks whole sentences, but gets into difficulties over words; he understands, in part, simple commands and requests; most of them, not at all or only partially. A *total aphasia* is rare,—*i.e.*, complete motor and sensory aphasia.

Word deafness disappears, as a rule, sooner than a motor disturbance.

The ability to use figures may be impaired or intact. A few cases seem to indicate that the memory for figures is connected in part with the right hemisphere. I treated a man with left-sided hemiplegia and hemianopsia, who found it difficult to count since the onset of the paralysis, because he could no longer conceive the figures.

A patient may be able to recite words which he has retained in his memory in a certain order, as the days of the week, the names of the months, the Lord's prayer, etc., although he cannot think of the words singly. He may not be able to give the name of the month in which he was born, although he knows it, and names it in running over the names of the months.

DYSLEXIA consists in the patient being able to read only a few words or sentences and then, fatigued and unwilling, unable to proceed farther. It has been assumed to be a partial alexia.

Causes of Aphasia.—Aphasia may be of a *functional* or *toxic* nature, or be produced by an *organic* disease.

The type of the functional form is the aphasia caused by *fright*. A severe psychic shock may not only momentarily paralyze speech, but produce an aphasia which lasts a long time. (See Hysteric Mutism.) The function of the speech centre may also be inhibited *reflexly*, especially in children. Aphasic conditions have been noticed in children which were due to the irritation of worms, and which were cured by a vermifuge. Temporary aphasia has also been observed in children after minor operations.

The aphasia accompanying, or ushering in, migraine attacks is probably produced by arterial spasm and faulty nutrition of the speech centres. It may be a transient symptom in conditions of *exhaustion*, loss of blood, and inanition. In the course of the acute *infectious fevers* (especially typhoid and pneumonia) it is more often observed. In pneumonia the aphasia generally develops on the second or third day, with apoplectic phenomena, has the character of motor aphasia, and may be combined with right-sided hemiplegia. It lasts some hours or days. It has also been noticed in *scarlatina*, generally in the later stage, after the nephritis. A toxic or infectious agent is probably at work. This is certain of *uremic* aphasia, which is, as a rule, a transitory disturbance. I have also seen an aphasia last for days in carcinoma, without being able to discover any cerebral alteration. A transitory aphasia has also been reported in santonin poisoning (Dunoyer); the same is said to occur occasionally at the height of an attack of gout.

Most cases of aphasia are, however, due to organic cerebral alterations, which attack either the speech centres directly, or the white matter just below the cortex. Lesions which lie deeper (internal capsule, central ganglia) do not, as a rule, produce aphasia, except by pressure upon the speech centres. The impulses from the speech centres can also be sent out by other tracts,—*e.g.*, through the corpus callosum to the right hemisphere and transmitted from here.

Hemorrhages, more often foci of *softening*, as they occur oftener in the cortex, are the principal causes of the aphasia. *Emboli* (or thrombosis) of the *middle cerebral* artery and its branches is a common cause. Hemorrhages into the external and internal capsules do not produce any lasting aphasia; they form only an indirect focal symptom, which, as a rule, soon passes away.

Abscess is not rarely a cause of sensory aphasia and its subdivisions (especially visual aphasia, as those coming from the left ear seem to favor the left temporal lobes); traumatic and metastatic abscesses may also cause motor aphasia.

Tumors in the speech centres, or in their vicinity, produce aphasia. *Encephalitis* and *tubercular meningitis* may also cause it. It has been noticed several times after gonorrhea, and was thought to be of embolic origin (Pitres, Bruns). It has been often traced to cranial *injuries*. In an interesting case in which the fragment lay upon the centre for speech, aphasia could be produced by pressure upon it (Dörrenberg).

In circumscribed cortical lesions it may be the only symptom of disease. Motor aphasia is often accompanied by *hemiplegia*, or by right faciobrachial monoplegia.

A lesion extending inwardly from the first temporal convolution often involves the posterior limb of the internal capsule and produces thereby, in addition to sensory aphasia, hemiplegia and hemianesthesia, and, by affecting the most posterior part of the internal capsule, or of the optic radiations, right hemianopsia. A focal softening not rarely injures the first left temporal convolution and the angular gyre at the same time; the word deafness is accompanied by alexia, generally right hemianopsia, etc.

Course and Prognosis.—Aphasia due to functional and toxic influences disappears, as a rule, quickly and completely. That is also the case in aphasia occurring at times after epileptic and paralytic attacks. If it is an indirect focal symptom, or caused by hemorrhage, it disappears generally in the course of some weeks or months. If the speech centre has been directly injured, and is not accessible to treatment, the prognosis can only be relatively favorable in juvenile and infantile cases. Recovery may occur in them, probably because the right hemisphere assumes the duties vicariously.

That the right hemisphere may assume the functions of speech, even in juvenile years, is shown by the following case, observed by me: A woman, who was right-handed originally, was forced, through an injury of her right hand, to use her left hand. It was in her seventeenth year that she became left-handed. When fifty-nine years old she became affected with a tumor of the right temporal lobe, and became aphasic.

The prognosis also depends upon the curability of the cause. Aphasia from traumata may often be cured by trephining, removal of splinters, or by the evacuation of a subdural hemorrhagic exudate. Syphilitic meningitis over the speech-centres and gummata in this region may become absorbed, and the speech defect disappear completely. Evacuation of abscesses of the left temporal lobe causes a disappearance of sensory or visual aphasia. The same results occur with the extirpation of a neoplasm which had injured, without destroying, the speech centres.

The prognosis is worse in cases with large foci of softening and with inoperable tumors. In the first case, the aphasia persists, though not always as a complete aphasia. The persons relearn a few words, sometimes a large number. The character of the aphasia has also something to do with the prognosis. A transcortical motor aphasia, for example, disappears more quickly and easily than a cortical one.

The treatment should be directed, in the first place, against the original trouble. Removal of bony splinters, foreign bodies, blood-clots, evacuation of abscesses, etc., may be necessary. If of syphilitic origin, an appropriate treatment should be instituted. Abscesses of otitic origin must be evacuated, operable tumors extirpated, etc. If the causal disease cannot be combated, some improvement may be secured by proper instruction in speaking and writing. This must be carried on systematically, in a manner similar to that of deaf and dumb persons, using the paths by which the sense impressions may still reach the speech centres. A person who can still read should be made to regain speech by reading; simple objects, as table, bread, etc., being, at the same time, held before him and their names written down. Writing may be utilized in a similar manner. If this is also lost, the tactile and muscular senses may be brought into action. Small solid letters are given him and he is required to put them together into words. Or his hand is led and a word written in this way until he has grasped it. The position of the mouth and lips for single letters must be learned by imitation. A teacher or relatives must be shown how to instruct the patient, as the physician is unable to give his time for such purposes.

To examine for aphasia, we must first satisfy ourselves that the patient hears. Next see if he understands words. Proceed from simple requests—"Touch your nose," "Let me see your tongue," etc.—to more complicated ones. Avoid gestures, as they can indicate to the patient what you are asking him. Then let him name objects held before him, or parts of the body, etc. If he cannot find the proper word, determine whether it is visual or motor aphasia. Next determine the nature of his speech, whether connected or not, whether he must deliberate long before finding a word, or confuses words, etc. Then find out whether he can repeat words after you, or can read or write. If he is unable to read, write a simple request and see if he understands the sense of it, even though he cannot read it aloud. Examine the power of simple copying, writing from dictation, and spontaneous writing. Avoid fatiguing or confusing the patient, and be positive that hysteria, dementia, etc., are not the cause of the aphasia.

DISEASES OF THE CEREBRAL MENINGES.

INFLAMMATIONS OF THE DURA MATER.

EXTERNAL PACHYMENINGITIS.—This is not an independent disease. It develops after diseases of the cranial bones, whether of traumatic origin or due to caries, lues, erysipelas, neoplasms, etc. The symptoms of pachymeningitis are often hidden by those of the original disturbance. Extradural suppuration from the ear may produce characteristic phenomena. (See chapter on cerebral abscess.) The affection has in general a surgical otologic interest.

Hemorrhagic internal pachymeningitis, or *hematoma of the dura mater*, is of more clinical interest, although rarely diagnosed during life.

Its *pathologic anatomy* is well known, though opinions differ as to the manner of its development.

According to Virchow, the inflammation of the dura mater is the primary occurrence. This causes the formation of a membranous deposit upon the inner surface of the dura, which is very vascular, and through rupture of these vessels hemorrhages occur. Others, as Huguenin, regard the hemorrhage as the primary alteration, while the connective-tissue membrane arises through the organization of the coagulum.

Post-mortem examinations reveal only slight alterations in mild cases or those which come early to autopsy: a delicate rose or grayish-red deposit upon the inner surface of the dura in the form of a veil-like membrane, which can easily be pulled off. This is generally perforated by punctate hemorrhages, and colored brown and yellow in some places from deposits of pigment from an older hemorrhage. The dura may be involved in small circumscribed regions, particularly over both parietal lobes, over the whole hemisphere, or even upon both sides of the brain. These deposits are also found in the middle and posterior cranial fossæ at the base of the brain.

The process occasionally becomes so intense that hard membranes, arranged in layers over each other, are formed, which cover the brain like a thick cap—between the dura and arachnoid—joining it to these membranes; while recent and old hemorrhages, which may become as large as a goose-egg, are formed in and between these deposits. The recent layers are soft and vascular; the older ones hard, firm, and but slightly hyperemic. The brain, especially the cortical part, is more or less atrophic.

Internal hemorrhagic pachymeningitis is mostly an *accidental* disease. The mild grades are seen in phthisis and chronic cardiac and kidney diseases. It is found particularly in cerebral diseases of a chronic inflammatory nature which result in an atrophy of this organ; in this manner it is commonly found in *paralytic dementia*, *chronic hereditary chorea*, and senile dementia. It may also, however, be a primary disease. It is relatively often observed in *chronic alcoholism*. It may also be noticed in those diseases produced by a hemorrhagic diathesis, pernicious anemia, leukemia, scurvy, Barlow's disease, purpura, etc. It rarely occurs from syphilis (v. Beck, Hahn). Finally, cranial injuries may produce it.

I have repeatedly seen in elderly persons suffering from severe constipation, producing over-exertion of the abdominal walls, symptoms which I thought were caused by a hemorrhagic pachymeningitis.

The disease may be symptomless, especially when the alterations are mild or accidental. In paralytic dementia, for instance, no symptoms of it may be evident. In some cases it is characterized by the symptoms of a cerebral affection, without their being distinct. We are justified in establishing a probable diagnosis when they occur under conditions in which hematoma of the dura mater often develops, as alcoholism, cranial injuries, etc. That the symptoms vary according to the seat and extent of the process is evident. After a prodromal stage of excitement with severe motor restlessness, which may resemble that of delirium tremens, or after intense headache, vomiting, and *unilateral* epileptic twitchings, the patient becomes comatose, and remains in this condition for days or weeks. The pulse is generally retarded, and at times irregular. An *apoplectic attack* occasionally ushers in the trouble. *Paralytic symptoms* only develop in the course of the coma, generally hemiparesis or monoparesis. The latter may develop gradually to a hemiparesis, and also spread to the other side. A contracture of the muscles of the other side may be combined with the *hemiparesis*. The *sensibility* is almost never impaired. *Aphasia* has been observed in only a few cases. *Unilateral* or *general* convulsions occur quite often.

Paralysis and spasms are often only transitory phenomena. *Choked disk* has been observed many times (due to a hemorrhagic exudate into the optic sheath), and several times only on the side opposite to the paralysis. *Conjugate deviation* of the head and eyes has been observed in a number of cases; at times nystagmus has been observed. The *pupils* are contracted at first; may be dilated later on the side corresponding to the paralysis. When the coma is absent or gone, a severe dull headache comes on, which may precede the coma.

Remissions and exacerbations occur very often; the coma may also clear up, and then deepen again. This is characteristic of chronic cases. The *temperature* gradually increases, and may reach 106° F. before the end of life. Subnormal temperature also occurs.

In comatose conditions it is difficult to distinguish the affection from meningitis. The stiffness of the neck is, however, absent, and the basal cranial nerves (except the optic) almost always spared. When commencing with an apoplectic attack, the further course, the irregularity of the paralytic symptoms, the intercurrent ocurrence of convulsions, and the choked disk of this disease tend to reveal this condition. It is not, however, possible to exclude with certainty a simple cerebral hemorrhage.

Treatment.—The therapeutic measures are limited to removal of all injurious things, application of an ice-bag, or bloodletting in plethoric persons. A purgative treatment is also advantageous.

TRAUMATIC HEMATOMA deserves special notice from both a symptomatologic and a therapeutic stand-point. Traumatic hemorrhages of the cerebral meninges are extradural or intradural. When caused by injury of the middle meningeal artery, it extends between the dura and the cranium. The intradural meningeal forms are generally due to lacerated veins of the pia mater. Hemorrhages may be caused by traumata which have not in any way divided the bones.

The patient awakens from the daze caused by the injury ; after a certain time, however, he again becomes unconscious.

These *free intervals* last from one or more hours to as many days, rarely longer.

If the interval lasts days or weeks (traumatic late apoplexy), it is generally not meningeal hemorrhage, but hemorrhages in the region of the aquæductus Sylvii and of the third and fourth ventricles, which are the result of a local softening (Duret, Bollinger, and others).

The patient generally becomes somnolent within some hours; the somnolence increases to coma, and the pulse becomes slow and tense. Unilateral, rarely general, motor symptoms of irritation, and paralytic symptoms, mostly of a partial hemiplegic character, also occur. Bilateral hemiplegia occasionally takes place. It is in traumatic hematoma that the so-called collateral hemiplegia has been often observed (Moullin, Ledderhose, and others). It has been explained above (page 438), that this has generally been the result of diagnostic errors. A bilateral hematoma has been seen several times. Aphasia is not an uncommon symptom. Hemianesthesia and hemianopsia are noticed more rarely. Fever, albuminuria, and glycosuria may occur.

The pupils on the side of the hemorrhage are often dilated; choked disk may also be confined to this side. It is always difficult to determine the location of the hemorrhage as long as the patient is comatose. Ortner believes that the condition of the respiratory musculature may aid materially in doubtful cases, as a unilateral paralysis of these (on the side opposite to the lesion) may be recognized in coma.

The patients generally die in the comatose state unless artificial help is given them ; the symptoms may, however, as I have many times observed, disappear partly or entirely. Some have attempted to decide by the clinical symptoms as to whether the hemorrhagic focus lies outside or within the dura (Brion, etc.), though they have not been entirely successful.

Lumbar puncture has been recommended and used for the diagnostic determination of the hematoma. A negative result can, however, as a rule, be expected, as a bloody fluid will only be found in the subarachnoid spaces of the spinal cord when the hemorrhage is located in the subarachnoid space.

Surgical treatment of traumatic hematoma of the dura mater has recently been of happy result (Macewen, Starr, Ceci, Wagner, Krönlein, Hahn, etc.). Even where no external injury revealed the seat of the hemorrhage, it has often been possible to determine this by means of the clinical symptoms, and by removal of the blood-clot, relieve the patient. Operative treatment is particularly indicated where the symptoms of brain pressure become dangerous and the local symptoms show no tendency to recede. I have seen such symptoms spontaneously recede (in one case even after an operation had been determined upon on account of the increasing brain pressure).

Hematomata of non-traumatic origin have been operated upon several times (Michaux, Jabouley).

ACUTE INFLAMMATIONS OF THE SOFT CEREBRAL MEMBRANES.

ACUTE (PURULENT) CEREBRAL MENINGITIS OR LEPTOMENINGITIS.

Acute inflammations of the soft cerebral membranes are generally of a diffuse nature. Though found especially on the base of the skull or on the convexity of the brain, it is not generally confined to any one of these areas. Most affections starting from the arachnoid and pia involve the brain, especially the cortex, so that in most cases we can speak of a *meningo-encephalitis.*

Etiology.—Purulent meningitis is caused by organized carriers of infection. Streptococci and staphylococci have been found in the exudate, but particularly a few micro-organisms whose specific influence is recognized. (Fraenkel's *pneumococcus* and Weichselbaum's meningococcus intracellularis.) The investigations of Leyden, Jaeger, Netter, Heubner, Fürbringer, and others seem to show that the latter is particularly the exciter of epidemic cerebrospinal meningitis, though Fraenkel's pneumococcus, Friedlaender's pneumobacillus, the typhoid bacillus, and the colon bacillus have been found, so that the question is not definitely settled. It has been possible to experimentally produce meningitis by the introduction of Fraenkel's pneumococcus and the meningococcus intracellularis (Netter, Foa, Fraenkel, H. Bruns).

The micro-organisms may reach the meninges from *neighboring foci* of *pus* or through the blood (and lymph) paths, from a *general infection* of the organism, or from *distant infectious foci.*

Injuries of the cranium—excluding traumata which directly involve the meninges—which are connected with the wounds may produce meningitis, the infectious germs causing a local suppuration which spreads to the membranes of the brain. A sinus thrombosis from the veins of the diploe may thus serve to transmit the infection.

Traumata which produce an opening into the cerebral canal, may also entail a purulent cerebrospinal meningitis. Meningitis has repeatedly followed *erysipelas* of the head. It has several times been ascribed to *actinomycosis.*

It is, however, particularly frequent after ear disease. *Purulent otitis* and *caries of the petrous bone* are the most important and frequent causes of purulent inflammation of the cerebral membranes. The purulent matter reaches this region through the thin tegmen tympani or through the top of the mastoid antrum, or is transmitted to the cerebral membranes through the nerve-sheaths of the acoustic and facial. Here also the venous and sinus thrombosis forms the connection between the otitic disease and the meningitis.

Suppuration from the *nasal cavity,* the *orbit,* frontal sinus, pharynx, etc., more rarely extends to the cerebral membranes. Intracranial purulent foci (cerebral abscess, extradural suppuration) may also extend to the meninges.

Epidemic cerebrospinal meningitis should be looked upon as a *primary infectious disease.* Purulent meningitis may also result from *pyemia, septicemia,* and the *acute infectious diseases* (pneumonia, typhoid, ulcerating endocarditis, variola, influenza, acute articular rheumatism, gonorrhea (?), etc.). Its connection with intestinal catarrh is still in doubt. It is highly improbable that purulent meningitis can result from a *sunstroke.* Nor is it proven that simple cerebral concussion or mental strain is able to cause this disease.

In many cases the etiology is obscure. There is a *sporadic* form of purulent inflammation of the cerebral meninges, which corresponds in its course to epidemic cerebrospinal meningitis, and probably has the same (infectious) origin.

It is advisable to treat of epidemic cerebrospinal meningitis and tubercular basilar meningitis separately, and therefore only the other forms of acute meningitis will be discussed here.

Anatomical.—Purulent—not tubercular—meningitis favors the convexity of the brain. Hyperemia of the pia first occurs, then follows a clouding of this membrane. The purulent deposit is at first found, particularly over the sulci, focal collections of pus develop, the foci coalesce, until a thick layer of greenish-yellow pus covers the membranes everywhere, especially on the convexity. The superficial layers of the cerebral cortex are also altered; serous infiltration, encephalitic processes, small hemorrhages, and purulent foci are found. Abscesses within the brain rarely occur. Foci of softening have also been observed. A seropurulent exudate into the ventricles, even a pronounced hydrocephalus, is a common condition in meningitis. It is mostly due to the fact that the openings communicating between the ventricles and the subarachnoidal spaces have been closed by the meningitic process. A purulent meningitis confined to the ventricles is said to occur in childhood; it is, however, no doubt, very rare. We find also a circumscribed purulent form of meningitis which only involves one hemisphere or even one lobe; an otogenous form of this character is particularly frequent.

The meningitis generally extends to the *spinal meninges*, though it is less marked here; it may even extend to the lowest portion of the cord. If the suppuration commences in the spinal meninges, it can quickly extend to the brain (Lichtheim). An otogenous meningitis may almost entirely limit itself to the spinal membranes.

F. Schultze showed that there is a cerebral disease developing from infection under the form of a meningitis, in which anatomic examinations reveal no, or only slight, meningeal alterations, but show round-cell proliferation in the brain and spinal cord. The meningitis may also be so foudroyant that it does not reach a purulent stage (Klebs, Leichtenstern).

Symptomatology.—Although the symptoms vary according to the seat, extent, and form of the meningitis, a clinical picture may still be drawn into which a typical case will always fit. The onset of the disease is often obscured from its occurring during the course of another affection, which in itself produces cerebral symptoms, as an infectious disease, cranial injury, etc. When distinct itself, the onset of the meningitis is characterized by severe, persistent, but exacerbating *headache*. This has a diffuse extension, but at times is felt mostly in the frontal and occipital regions. This is followed in a few days by a dulling of the sensorium; the patient becomes dazed, delirious in sleep, afterwards constantly; or a condition of *somnolence* varies with one of delirium. It is characteristic that the headache continues during the delirium, so that the patient in his stupor and hebetude occasionally cries out on acount of the pain. Even in sopor, he grasps his head, makes painful grimaces, especially when it is sought to move the head passively.

As long as his sensorium is free he complains of *vertigo*, and especially of sensitiveness to light and noise. This *hyperesthesia* may reveal itself in the stage of stupor by the shrinking of the patient from touch, noises, etc.

Vomiting is often, if not always, present, and has all the signs of a cerebral vomit.

Fever is present, as a rule, from the beginning. The temperature may rise in the first few days to 104° F. and remain at this height until death comes on, though irregular variations usually occur. Ante mortem another rise, even to 108° F., occurs, or the temperature becomes subnormal. Chills may manifest themselves at the beginning of or during the disease.

The *pulse* is generally frequent, though it may not be increased. A slowing of the pulse to fifty beats a minute, or lower, may occur at the beginning.

One of the most important symptoms of meningitis is the rigidity and retraction of the muscles of the back of the neck. It is probably due to meningitis of the posterior cranial fossa, and is, therefore, a constant symptom of basal meningitis. A certain degree of rigidity is also found in the rest of the musculature of the body. Place the patient in a sitting position, so that the legs hang out of the bed—a muscular tension of the flexors of the knee is distinctly noticeable; the leg cannot be completely extended (Kernig). The abdominal muscles are often so contracted as to be as hard as a board, and the abdomen is trough-like in shape.

Hyperesthesia of the skin and muscles is frequently present. Mere touch will call forth lively reflexes. Percussion of the lumbar muscles produces a spastic retraction of the spinal column (spinal phenomenon). The vasomotor excitability is also exaggerated, stimulation of the skin producing an intense and persistent redness, though this symptom is in no way characteristic. Herpes and other skin affections rarely occur in non-epidemic meningitis.

Symptoms pointing to involvement of the *brain* and of the *cranial nerves* also occur. The latter are the more important and constant, and occur particularly when the meningitis extends over the base of the brain. The nerves for the ocular muscles, the facial, and the optic are especially involved.

Pupillary differences are often found early in the disease; the pupils are *generally* contracted at first, later dilated; *pupillary rigidity* also occurs. The mydriasis may to-day be noticed in the left, to-morrow in the right eye. *Ptosis* and paralysis of one or more muscles of the eyeball, with a corresponding *strabismus*, are often noticed; less often, nystagmus. A noteworthy, but by no means constant symptom, is *optic neuritis*, which is often only partial, and which rarely develops to a complete choked disk. Vision is rarely decreased in power; its examination is difficult or not at all possible. (Consult the next chapter for other ocular complications of purulent meningitis.)

Paresis of the facial is present rather often. It is generally preceded by sharp contractions in the facial muscles, or they continue after the paresis has appeared. It is mostly combined with a *reaction of degeneration*, though I have been able to detect this in only one case, as the disease generally ends in death before this alteration can mature. *Trismus* is occasionally noticed. Symptoms of irritation and paralysis of other cranial nerves have been observed in only a few cases.

The involvement of the cerebral cortex manifests itself by many symptoms. Fleeting *twitchings* in the extremities, *unilateral* and *general convulsions*, also contractures on one side of the body, are the most frequent of the early symptoms. *Monoplegia* or *hemiplegia*—more rarely only a central facial paralysis (limited to the lower branches)—come on later, or are seen from the beginning.

Aphasia is frequently noticed in tubercular basilar meningitis, rarely in the other forms, as, for example, in a case of Kuhn's, of otitic meningitis. Hemianopsia has hardly been diagnosed with certainty in cases of acute meningitis.

Unilateral hyperesthesia is seen at times; hemianesthesia, almost never.

The deep reflexes are generally increased at first, but tend to decrease later on. This should not of itself be ascribed to the spinal meningitis, as the high fever may cause the knee-reflexes to disappear. The fact that it is only absent for a time in some cases indicates this.

Constipation is frequent. In the stage of sopor, *retention of urine*, more rarely *incontinence*, is present. The scanty urine may contain some albumen. Glycosuria is rare. The strength fails rapidly. *Decubitus* occurs, especially in the last stage.

We find at this stage deep coma with lost reflexes. The pulse becomes small and very frequent; the respiration, which may have been irregular from the beginning, becomes rapid, superficial, or irregular, or Cheyne-Stokes breathing may come on. Death occurs in deep coma, at times in convulsions.

The course of the disease is always *acute*, and may be so violent as to cause death in forty-eight hours. It lasts, as a rule, one or two weeks, rarely longer. As the symptoms of irritation occur in the beginning and the paralytic symptoms later on, some have endeavored to distinguish two stages, but this is hardly possible, as the paralytic symptoms, for example, may occur in the beginning.

The clinical picture of purulent meningitis is *very variable*, and may deviate completely from the above description. When occurring in the course of another disease, it may long remain latent (Huguenin, Fraenkel, Jansen, and others). In some cases fever is absent; in others the rigidity of the back of the neck is missed. Very rarely, however, does the sensorium remain free to the last. The disease may take weeks to develop.

Attempts to refer the different symptom-complexes in meningitis to differences in the micro-organisms causing the disease have not been very satisfactory.

Sinus thrombosis and *cerebral abscess* are the most frequent *complications* of meningitis.

The prognosis of purulent meningitis is very bad. Recovery is rare. Gowers cites two cases of meningitis, following upon puerperal sepsis, which ran a favorable course. Recovery should not be expected after coma has set in. Predictions should, however, be cautiously made, as long as the diagnosis is not entirely certain.

Differential Diagnosis.—Typical cases are easily diagnosed. A diagnosis may, however, be very difficult under certain circumstances.

It is not uncommon, for instance, for an acute infectious disease, which is ushered in by cerebral symptoms, particularly *pneumonia* and *typhoid*, to resemble meningitis. The headache, delirium, stupor, fever, and perhaps vomiting, are symptoms which tend to cause this error. A diagnosis of meningitis should not, therefore, in doubtful cases be made upon these symptoms alone. Special caution is necessary with children, as some children become delirious and are stricken with convulsions upon every rise of temperature, so that even a simple angina may at first be thought to be meningitis. A closer examination of the lungs will guard against confusing it with pneumonia. The detection of a splenic tumor, roseola, the characteristic stools (generally obstipation in meningitis), the characteristic temperature, etc., should make typhoid fever recognizable. In addition, typhoid develops *gradatim*, meningitis acutely and—excluding the tubercular form—without prodromes. Cases of this kind should only be diagnosed as meningitis when the pathognomonic phenomena, rigidity of the back of the neck (may also be a prodrome of typhoid), paralytic symptoms in the area innervated by the cranial nerves, optic neuritis, cortical phenomena, etc., are present. The continuance of the headache during the delirium and somnolence is suspicious, and indicates meningitis. Retardation of the pulse without any increase in temperature must also give rise to a suspicion of cerebral trouble. *Septicemia* and *pyemia* are likewise accompanied by cerebral phenomena which may simulate the picture of meningitis, though the signs of involvement of the cranial nerves and the stiffness of the back of the neck are missing during their further course. Swelling of the joints, phlegmonous processes, hemorrhages in the skin and retina, repeated chills, etc., suggest septicemia. A careful examination will likewise reveal a source for the septicemic process. A blood examination would probably only in rare cases, by revealing the presence of pyogenic micro-organisms, make a diagnosis certain.

An *acute purulent otitis* also presents cerebral symptoms at times, especially in childhood, corresponding in many respects to those of meningitis. They are headache, vertigo, and stupor. Delirium and general convulsions may also occur with children, and, though difficult to understand, optic neuritis was observed several times. All these phenomena may disappear with the removal of the pus or the disappearance of the otitis. The relations between ear diseases and cerebral disorders which are similar to meningitis, but which may end in complete or partial recovery (with persistent visual disorder, even with optic atrophy), is not yet completely explained. It appears that the so-called serous meningitis is often a cause of the symptoms. Extradural abscesses of the posterior cranial fossa may also produce a clinical picture resembling meningitis in many ways. (See chapter on Cerebral Abscess.)

It is probably always possible to diagnose uremia from an examination of the urine, the edema, etc.; albuminuria may also occur in meningitis, but the casts, etc., are absent; in doubtful cases the clinical course furnishes an aid to diagnosis.

Syphilitic meningitis may run an acute course and be combined with a slight elevation of temperature, but this is rare. In such a case, in which ptosis, pupillary differences, rigidity of the back of the neck, and stupor were the chief symptoms, recovery quickly followed the administration of potassium iodide.

A number of conditions have been described under the names of *pseudomeningitis*, *méningisme*, etc., by Kohts, Dupré, Seitz, Lépine, Kraunhals, and others. They consisted of symptoms and symptom-complexes very similar to meningitis, while the termination in recovery or the autopsy teaches that no purulent meningeal inflammation was present, and that either no alteration or only edema, hyperemia, or perhaps a serous exudate was the basis of the condition. In some of these cases a toxic cerebral disease was probably the cause, in others the course was too violent for it to reach the purulent stage; finally, the so-called serous meningitis (see chapter on Acquired Hydrocephalus) may produce a clinical picture allied to that of purulent meningitis. These observations show the caution necessary in making a diagnosis of purulent meningitis. Especially where the cerebral symptoms occur in the course of an acute infectious disease (particularly *pneumonia* and *influenza*) or after these diseases, and when they deviate from the type of meningitis described above, is this caution in place.

Non-purulent meningitis may be due to otitis (Levi, Joel, Kretschmar, and others).

Meningitis is apt to be confused with *delirium tremens* in alcoholics. Meningitis may here produce a real alcoholic delirium, which will completely shroud the symptoms of the meningitis. In the delirium, the rigidity of the back of the neck, the intense headache, and especially the focal symptoms are absent. When the individual is brought to the hospital in the last stage, or when the meningitis takes a furibund course, it may not be diagnosed.

Some of the meningitic symptoms, somnolence, convulsions, trismus, etc., may occur in children weakened by gastro-intestinal catarrh.

In those conditions styled *encephaloid*, the fever, focal symptoms, and disease of the optic nerve are missing, and, in addition, the etiology, the depressed fontanelles, and the accessory symptoms will establish the diagnosis.

Finally, *hysteria* may cause diagnostic errors, in that it may produce delirium, sopor, and also rigidity of the cervical muscles. I was only lately called to see a patient whose head had been shaven and antimonial ointment rubbed in on account of these symptoms. A few hours afterwards she was able to leave her bed. In taking the history the genesis is to be inquired into above everything else. The known causes of meningitis are absent; on the contrary, psychic excitation has in most cases immediately preceded the other symptoms. A rise of temperature is almost always absent in these hysteric attacks; at least it does not correspond to the fever of meningitis. The pulse is neither slowed nor arrhythmical; the pupils are equally wide and react normally; optic neuritis, facial paresis, etc., are always absent. The eyes are generally held tightly closed, etc.

Acute lethal hysteria is such a rare affection that the differential diagnosis need hardly be given.

In recent years *lumbar puncture* has been used as an aid to diagnosis. This method, established by Quincke in 1891, consists in obtaining a small quantity of the cerebrospinal fluid contained in the subarachnoid spaces of the spinal cord by puncture of the spinal canal, and subjecting it to a physical, chemical, microscopical, and bacteriological examination.

Quincke recommended the following method: The patient is to be placed upon his left side, with his lumbar vertebral column strongly inclined forward. Find the space between the third and fourth or between the fourth and fifth lumbar vertebræ, and pierce the skin,—in children in the median line, in adults about one centimetre laterally from it, at the height of the lower border of the spinous process, or at the height of its lower third. Introduce the needle somewhat upward and medianward and inward until, from the cessation of all resistance, we recognize that the point has penetrated into the subarachnoid space.

To measure the pressure, a glass tube of narrow bore or a mercury manometer is connected with the canula by means of a cone and rubber tubing.

The method and the apparatus have been variously modified. Some select the space between the arches of the second and third lumbar vertebræ; and Chipault (afterwards Fürbringer), the sacrolumbar hiatus, the region between the last lumbar vertebra and the upper border of the sacrum, as a place for inserting the needle. It depends upon the individual whether we should pierce in the middle line or somewhat laterally from it, at the height of the lower border of the spinous process or higher; in general it is advisable to follow Quincke's directions, and only modify his method when we strike against bony resistance. According to the thickness of the areolar tissue and the other parts which the needle must pass through, it is necessary to insert it more or less deeply. The difference in individuals varies from two to seven centimetres.

Quincke uses a canula of one-half to one millimetre bore, with a mandrin attached. Braun prefers the hollow needle. Quincke's apparatus has been modified by Krönig. In the first the pressure can only be measured after the flow of some cubic centimetres of liquid, while Krönig avoids this defect by a measuring apparatus consisting of capillary tubes which permit the measurement of the pressure from the outset. A mercury manometer can be dispensed with; a U-shaped glass tube, whose ascending limb must be of a satisfactory length (about one-half to one metre) is sufficient. It is not necessary or desirable previously to fill this tube with a sterilized salt solution (Stadelmann).

The removal of a small quantity of cerebrospinal liquor is sufficient for diagnostic purposes. The pressure must be controlled, and in a rapid sinking the discharge must be checked. Aspiration should be avoided. It need not be said that this small operation must be done antiseptically. Narcosis is not necessary; local anesthesia is sufficient, if any is needed.

The normal pressure is, according to Quincke, forty to sixty millimetres water, though he only considers an increase to over one hundred and fifty millimetres to be pathologic. Krönig gives much higher figures. Under pathologic conditions the pressure may increase to seven hundred millimetres or more. We can also in general speak of an increased pressure when the liquor flows in a full stream.

Normal cerebrospinal liquor is clear, contains only two- to five-tenths per cent. of albumin and a little cellular matter. Pathologic conditions are characterized by the following alterations: increased quantity, increased pressure, cloudiness of the liquor, presence of blood, fibrin clots, or pus, increase in the quantity of albumin (to seven-tenths per cent. or more), coagulability, presence of micro-organisms in the liquor. These factors are of unequal value. The increase of quantity and pressure occurs under various relations, especially in cerebral tumor, sinus thrombosis,(?) hydrocephalus, or serous meningitis, the different forms of meningitis, and other conditions (uremia, chlorosis). It is not a constant phenomenon of any of these affections. Especially in meningitis is the increase in pressure and quantity often absent. An examination in this direction will fail when communication between the ventricles and the arachnoid spaces of the brain and cord is prevented on account of adhesions, or displacement of the foramen of Magendie, etc. The albumin is increased in inflammatory conditions and congestive hydrocephalus (tumor); but, on the whole, the diagnostic importance of this symptom is slight.

The presence of *bloody* constituents may be due to piercing of a vessel during the puncture, but is particularly caused by hemorrhages into the meninges of the brain and cord, as well as by hemorrhage into the ventricles.

The liquor is *clouded* in the first place by *purulent* meningitis. It may also be cloudy in the tubercular form (rarely purulent or bloody). A *purulent* condition is, however, especially characteristic of the different forms of purulent meningitis. If pus is absent, the large number of leucocytes may reveal a purulent inflammation (characteristic tumor-cells may perhaps be found in malignant tumors).

The bacteriologic examination is very important; it must be carried out with hanging drops and stained preparations, and by means of cultures. In purulent meningitis *streptococci* and *staphylococci*, and particularly the *pneumococcus*, as well as the *meningococcus intracellularis* (Jaeger, Weichselbaum), have been found (Lichtheim, Heubner, Fürbringer, and others).

In the epidemic form the latter and also Fraenkel's coccus have been noticed in particular. In tubercular meningitis, *tubercle bacilli* have been found, especially in the clots (it may be necessary to centrifugalize), according to some in most cases (Fürbringer, seventy per cent.), according to others in only a slight percentage.

These results are especially important in that they are absent in symptomatically allied diseases (serous meningitis, tumor, hydrocephalus, cerebral abscess, etc.).

The diagnostic importance of these criteria is, however, considerably decreased on account of their inconstancy. The transudate, for instance, may be clear in purulent meningitis, free from leucocytes and characteristic micro-organisms (Lichtheim, Stadelmann, Oppenheim). This is true of all cases in which the communication between the brain and the subarachnoid spaces of the spinal cord has been interrupted. The exudate may also be plastic, thick, and gelatinous, so that the result of the puncture becomes negative. Or the canula reaches the subdural space by reason of the arachnoid and pia having grown together (Stadelmann).

If a large quantity of clear fluid is discharged, it is very improbable that a universal purulent meningitis is present. On the other hand, puncture in some cases of cerebral abscess with circumscribed purulent meningitis has given a cloudy liquor (Stadelmann). Other unfavorable results during the operation are: The needle may pass in between the dura and the bone (subdural space), it may be clogged by fibrin clots, etc. The change in pressure produced by the removal of the liquor may increase some of the symptoms, especially the pain in the head. If a root of the cauda equina is pierced, there may be pain in the corresponding nerve-area. It is not impossible for a circumscribed meningitis to be converted into a universal one through the puncture, or for a cerebral abscess to break through (Stadelmann, Oppenheim). Several times has death resulted soon after the operation, especially in cerebral or cerebellar tumor, and also in meningitis, aneurisms, etc.

As, therefore, the danger is not inconsiderable, and as diagnostic conclusions can only be drawn with caution, puncture should not be used *ad libitum*, but only where the diagnosis is doubtful, and where it is to the interest of the patient definitely to determine it. For the therapeutic value of this operation, see below.

Treatment.—As soon as there is any suspicion of meningitis, absolute rest in bed should be ordered. The head should lie high and the neck be free. All excitement must be avoided, also every noise; even light may be painful to the patient.

Purgation by means of calomel and similar remedies is desirable.

Experience has also shown the benefit obtained by applying an icebag to the shorn head. In strong individuals, a local *bloodletting* by means of leeches applied to the mastoid region is in place, though much blood should not be drawn. Severe headaches should be combated with morphine, which drug should also be used for severe vomiting and furibund delirium.

Some physicians recommend inunctions of the ointment of the tartrate of antimony upon the bare head; the use of a fly blister in the occipital region is occasionally palliative. Energetic measures of this kind are not to be advised, as they bother the patient and are of doubtful utility. Diuretics and the iodide of potassium and mercury have warm advocates; their use is, however, of doubtful benefit. *Cold douching* of the head, the patient sitting in a lukewarm bath, is recommended for the sopor.

It is important to seek after the source of the disease and to *remove every accumulation of pus near the cranium, and also inaccessible extradural abscesses.* Paracentesis of the tympanum, *trephining of the mastoid process*, or incision of a cerebral abscess may be necessary. Many cases of incipient meningitis have been prevented in this manner. The aurist has many opportunities for preventing meningitis and combating incipient cases. A rational treatment of cranial injuries is also of prophylactic value.

There is still much uncertainty regarding *operative* treatment of this disease. Some cases have been cured by ventricular puncture (v. Beck), others, in which the suppuration started in the ear, by incision of the dura mater and evacuation of the exudate found in the meninges (Macewen). Excluding traumatic meningitis of the convexity of the skull, we are far from considering these operations as being indicated and advisable.

Lumbar puncture has often been used in these cases (Lichtheim, Quincke, Heubner, Fürbringer, and others). Only in a few cases of purulent meningitis, as in those of Ricken and Jansen, has it been of much benefit. Further observations should, however, be made, especially when the signs of cerebral pressure are marked. The value, technique, and dangers of this treatment have been discussed above. The value of incision of the spinal dura as recommended by Quincke has not yet been sufficiently determined. Trephining of the vertebral column with incision of the dura and drainage (Paget) has been used several times, without any noticeable result.

EPIDEMIC CEREBROSPINAL MENINGITIS.

Purulent cerebrospinal meningitis quite often becomes epidemic. It is no doubt a *hetero-infectious disease.* Fraenkel's pneumococcus (Leyden) and especially the diplococcus intracellularis (Yeager, Weichselbaum, Heubner, and others) have been found in these cases.

As this latter micro-organism has been discovered once in tubercular meningitis and in the nasal mucous membranes of healthy children, it shows that caution is necessary in regarding its presence as being of specific importance. It is, therefore, not settled whether this disease is caused by a definite micro-organism.

The epidemics occur generally in winter and spring, though they may come on in the warmer season. Direct transference of the disease does not seem to occur. *Children* and *youthful* individuals, in some epidemics exclusively children, were particularly attacked. In one statistic, eighty-two per cent. of all cases were in children of from one to ten years of age. Persons over thirty are rarely affected.

There are *sporadic* cases in which the clinical picture resembles that of the epidemic form in all respects, and it is extremely probable that the same micro-organisms are at work. The fact that several persons became sick in the same city, in the same street, without our being able to speak of an epidemic, also indicates this. The poorer classes and those living in prisons, asylums, etc., are particularly in danger. Its occurrence does not immunize the individual against future attacks. Traumata may act as an inciting cause.

Symptoms.—This disease has a very variable clinical picture. Typical cases present the following symptoms : The onset is abrupt, or for some hours or even one or two days previous, we find the following prodromes : general indisposition, chills, restlessness, slight pain in the back and head, and drawing pains in the extremities.

The disease itself now commences with severe *headache, vomiting, vertigo*, sensitiveness to light and noise, and often, also, with chills, and, more rarely (in children generally), with *general convulsions*.

The patient becomes restless and sleepless ; the sensorium, however, still remains free. Immediately, or after one or two days, the ominous *rigidity of the back of the neck*, the *nuchal stiffness*, betrays the character of the disease.

The temperature is increased, does not exceed 102° F. at the beginning, but varies greatly, as does the irregular and rapid pulse. Enlargement of the spleen is generally present, but not always detectible.

In the next few days the headache increases ; the fever continues or exacerbates ; the patient becomes wildly delirious ; the muscular rigidity extends to the muscles of the maxillary, spinal, and truncal regions, and to those of the extremities.

The head is bent backward almost at a right angle ; trismus and opisthotonos occur. The abdomen is frequently drawn inward. Every contact with the skin, every pressure upon the muscles, especially attempts at active and passive movements, produce severe pain, which reveals itself by facial contortions, even during the delirium and the stupor which have come on. Obstipation and, generally, retention, later incontinence, of the urine occur. The quantity of urine is sometimes increased. Albuminuria and mellituria occur now and then.

If the disease takes an unfavorable course, coma comes on during or at the end of the first week, and paralytic symptoms appear. The face becomes pale and pinched, the pupils are generally wide and fixed, and the ophthalmoscope reveals a beginning *optic neuritis* ; strabismus, oculomotor paralysis, and facial paresis are not rarely observed. If coma is absent, or if the sensorium clears up, some *deafness* may be noticed.

Conjunctivitis, chemosis, keratitis, and especially purulent iridochoroiditis, may occur during the course of a meningitis.

The chemosis may be a result of inflammation of the orbital cellular tissue, which Axenfeld was able to show to be due to the entrance of pneumococci. The keratitis may be of a neuroparalytic character,—*i.e.*, a result of the involvement of the trigeminal. Amaurosis results from direct involvement of the optic nerve (descending perineuritis, optic neuritis) ; it may, however, also exist with normal ophthalmoscopic conditions, and be a result of injury to the basal visual structures (chiasm, tract), or it may be of cortical origin (Uhthoff). Whether the optic neuritis is due to the micro-organisms themselves (Axenfeld was able to trace them to the optic sheath) or to their metabolic products is not known. Purulent iridochoroiditis seems to be mostly a metastatic process (Oeller, Axenfeld); it leads to amaurosis. Axenfeld calls this condition meningitis ophthalmia. Panophthalmitis with perforation is rare.

General or partial convulsions are not so infrequent in the first stage, and may occur repeatedly. A *monoplegia* or *hemiplegia* shows itself when not overshadowed by loss of consciousness. Aphasia is rarely present. The phenomena of a spinal paralysis appear in many cases: *paraplegia*, Westphal's sign,[1] vesical paralysis, girdle pains, etc., which are naturally distinctly noticed when the sensorium clears up. Paralysis of the nuchal muscles occurs occasionally.

Efflorescences on the skin form early and often,—*erythema*, *urticaria*, *roseola*, *miliaria*, *purpura*, etc. Herpes appears, as a rule, even in the first days, and is an important diagnostic point. The vesicles are found particularly on the lips, sometimes also on the face, ears, and in symmetrical arrangement on the extremities. In some epidemics herpes is present in almost every case. It lasts, as a rule, some days, and then dries up. Micro-organisms have been found in the vesicles.

Simple or purulent articular exudates are sometimes found.

In fatal cases the coma continues, the patient continually emaciates, the urine and feces are evacuated involuntarily, and profuse perspiration and meteorism may come on near the end of life; the pulse becomes very small and frequent, and intermits; the temperature increases to 108° F. and over, or becomes subnormal; and death occurs at the end of the second or beginning of the third week. In not a few cases death results much later, after weeks or months. In such cases the disease either took a *protracted* course, or death was due to decubitus, exhaustion, etc.

In favorable cases the symptoms are generally slight from the first; the coma particularly is incomplete or absent, and the temperature soon falls or becomes remittent. In the course of the second week the symptoms subside, the restlessness decreases, appetite returns, and the patient becomes convalescent, though unfortunately still threatened with *relapses*. It may take a very protracted course, and sometimes a chronic condition follows, due to chronic meningitis, hydrocephalus, etc., in which the symptoms described continue with slight intensity, and paralytic conditions, which will later be described, become prominent symptoms. Here, also, death may result after a long time.

---

[1] The deep reflexes are at first generally increased, and may remain increased in the further course, but disappear more often, at times only for a short period.

Of the *modifications* of this disease the following are of special interest:

1. *Fulminant epidemic cerebrospinal meningitis.* An individual, healthy until then, becomes suddenly ill. Chills, headache, vomiting, delirium, frenzy or coma, nuchal rigidity, paralysis, etc., develop within a few hours, and death results just as quickly.

2. In contrast to this form are the mild, rudimentary, or *abortive forms*, which can only be recognized with certainty at the time of an epidemic. The individual complains of headache and backache; is somewhat restless and dazed; vomiting may occur, also local or general spasms, a slight nuchal rigidity, etc. These phenomena last for from one or more days to one or two weeks, to end finally in recovery. Sometimes only a slight pain in the head and nucha, with a general indisposition, is present, the relation to the epidemic form being a doubtful one.

3. In a third category of cases the disease takes a protracted course and lasts for months. The fever may also be *intermittent* and may represent a quotidian or tertian type; in the intervals the patient is without fever, and the other symptoms also disappear, to exacerbate with the fever. These intermissions only appeared in some cases in the convalescent stage.

Recovery from epidemic cerebrospinal meningitis is sometimes incomplete, certain symptoms remaining a long time or constantly. To these belong a tendency to *headache*, and also *tinnitus aurium*, *strabismus*, *blindness* (*optic atrophy* from *neuritis* or *phthisis bulbi*, or, very rarely, from disease of the visual centres), and very often *deafness*. The deafness occurs in the first two or three weeks of the disease, more rarely in convalescence, and still more seldom after months. If it affects early childhood, deafness and dumbness result; thus, the deafness and dumbness acquired in childhood is often a residuum of epidemic cerebrospinal meningitis. Even the rudimentary forms of this disease may result in permanent deafness. In most cases a disorder of gait occurs at the same time, due to faulty incoördination. The patient walks unsteadily, sways, and easily reels and falls. It is particularly marked in children, but tends to disappear in the course of some months or years.

Meningitis more rarely leaves behind it a paraplegia or paraparesis of a spinal type, which lasts a long time or becomes a chronic condition.

In one of my cases the following symptoms remained after an attack of cerebrospinal meningitis: occipital pain; vertigo, especially upon inclining the head backward; dysphasia, nasal speech; paresis and contracture of the left facial; paresis, contracture, and ataxia of the right extremities; impaired hearing; acceleration of the pulse; inclination to fall backward upon closing the eyes; and rhythmical twitchings of the velum palatinum and of the laryngeal muscles. I referred these phenomena to a chronic meningitis of the posterior cranial fossa and an encephalitic focus in the left pontile region.

Weakness of the memory may also be a residuum of this disease.

Pathologic Anatomy.—In cases progressing rapidly to death, hyperemia of the soft cerebral membranes and serofibrinous exudates may be the only alterations, or the result of the examination may even be negative. Generally, however, a fibrinopurulent or purulent exudate in the pial meshes, on the apex and especially at the base of the brain, on the upper surface of the cerebellum, and on the spinal meninges in their whole extent, or particularly at the height of the lumbar prominence, is found. In the spinal cord it extends especially along the posterior surface. The ventricles contain a cloudy, purulent liquid. If of long duration, a marked hydrocephalus may develop, and the exudate in the pial meshes undergo a caseous degeneration (v. Ziemssen). Inflammatory foci and small abscesses may also be present in the substance of the brain (Klebs, v. Strümpell).

The cranial nerves are partly imbedded in and infiltrated with the exudate; the process may continue along the acoustic to the labyrinth, and produce a purulent inflammation there; it is, however, not improbable that the infectious material acts, from the beginning, upon the cerebral membranes and the inner ear at the same time. Otitis media with perforation of the tympanum has been occasionally observed. Iridochoroiditis and panophthalmitis also occur.

Of the alterations in other organs, bronchopneumonia, pleuritis, and articular diseases should be mentioned.

Differential Diagnosis.—The epidemic occurrence of the disease, the herpes, the early development of the opisthotonos and of the spinal meningitic symptoms, generally allow us to differentiate this form easily from the others, especially the tubercular. The herpes, in particular, seems to be an important point, as it rarely occurs in tubercular meningitis.

The detection of tuberculosis of other organs and the results of spinal puncture (see page 478) should be valued.

Typhoid fever differs from epidemic meningitis in the characteristic temperature, the character of the stools, the meteorism, etc. The herpes is also absent here; and, though nuchal rigidity occasionally occurs, it does not extend to the muscles of the trunk and extremities. Finally, the results of lumbar puncture may decide the question.

Meningitis has only the muscular rigor in common with *tetanus*, so that they should easily be differentiated.

Uremic coma may produce many of the symptoms of meningitis (convulsions, vomiting, even nuchal rigidity), but the fever, herpes, hyperesthesia, etc., are lacking. A urinary examination is not always decisive, as albuminuria and urinary casts may occur in epidemic meningitis.

If epidemic meningitis commences with swelling of the joints, it may simulate the picture of an acute articular rheumatism; if, however, the whole symptom-complex and course are observed, error is hardly possible.

Hysteric nuchal rigidity, which is not uncommonly observed during an epidemic, presents neither the rise in temperature nor the signs of paralysis of the cranial nerves; on the other hand, hysteric symptoms are present, and it is almost always possible to establish the psychogenous nature of the opisthotonos.

The prognosis is always grave: life is very much in danger. The mortality in the different epidemics varied between twenty and eighty per cent., with an average of from forty to fifty per cent. At the end of an epidemic, the milder cases predominate.

The chances for recovery are very slight when the course is stormy, fever high, and the coma of early onset. The prognosis is better in cases of a mild course from the first, especially when the coma only appears later, is slight, or is entirely absent. If improvement does not occur in the course of the second or third week, recovery is improbable.

The prognosis is clouded by the circumstance that the meningitis rather often combines itself with other infectious diseases, especially with croupous pneumonia, rarely with scarlatina, endocarditis, pericarditis, etc.

Of the resulting diseases, the blindness and deafness have, on the whole, a bad prognosis. If the latter does not recede in the first three months after the outbreak of the disease, it will continue for life.

Treatment.—The directions laid down for the other forms of meningitis apply here also.

The application of an ice-bag or slight bloodletting and purgation are to be recommended. Potassium iodide, sodium salicylate, and digitalis have been used without much result. Hot baths have lately been recommended. In severe cases, morphine injections can hardly be avoided to combat the severe pain, hyperesthesia, and restlessness. Concerning the surgical treatment, consult the preceding chapter. Several times recovery has occurred after (also as a result of ?) the repeated use of spinal puncture.

We have no remedies to cure the residual symptoms. Sweat baths are recommended.

TUBERCULAR MENINGITIS; TUBERCULAR BASILAR MENINGITIS.

Tubercular meningitis is due to an invasion by tubercle bacilli of the cerebral membranes, and is always a secondary disease. Primary tuberculosis has its seat in the lungs, pleura, caseous bronchial, mediastinal, and mesenteric glands, in the bones or joints, in the testicles, or in the urogenital region. If a tubercular focus is not found upon autopsy, it may be hidden anywhere, and simply not be discovered; a caries of the inner ear, of the sphenoid bone, or a caseous lymph gland being particularly liable to be overlooked.

It is not settled in what way the bacilli reach the meninges. They can enter the arteries and be carried to the brain with the arterial blood. Caseous material may, for example, be taken up directly by the pulmonary veins. The extension of meningeal tuberculosis to the vascular area of a meningeal artery indicates an *embolic* origin. Strümpell believes that the virus passes from the lymph sheaths of the nerves to the arachnoidal pockets of the spinal cord, and from here reaches the base of the brain. Leube is also inclined in some cases to regard the disease as having arisen in this manner.

The conditions which influence the infectious material to favor the meninges as a seat for propagation and extension are not known. The brains of the young are predisposed, as tubercular meningitis occurs for the most part in *childhood*, between the second and fourteenth years of life. Individuals of from fifteen to thirty-five years of age are still affected; after the fortieth year it is very rare. It is possible that the life processes in a child's brain, which we look upon as being combined with rapid metabolism and a correspondingly increased conveyance of nourishment, are to a great extent responsible. When the predisposition exists, a *trauma*, mental strain, and particularly an *acute infectious disease* may cause it. Tubercular children, after an attack of measles and whooping-cough, are noticeably often affected by tubercular meningitis. Alcoholism is also regarded as a predisposing agent (Boix).

Pathologic Anatomy.—The eruption of miliary tubercles in the soft meninges, and inflammatory alterations of these membranes, compose the pathologico-anatomic basis of this disease. The serofibrinous, gelatinous, rarely purulent exudate invades principally the base of the brain near the chiasm, between it and the cerebral peduncles, and extends from here into the Sylvian fossæ, and backward to the basal surface of the oblongata, cerebellum, etc., and in most cases to the membranes of the spinal cord. The convexity is less often involved; sometimes here also a cloudiness and infiltration of the pia, especially over the sulci and along the blood-vessels, is seen, accompanied by band-like strata of the exudate. More rarely, extensive superficial foci of a tubercular inflammation with caseation are found in this region.

The miliary tubercles which are imbedded in the exudate, and appear also in places free from inflammation, are fine, grayish-white, translucent nodules. There may be few or many, and like the exudate they are grouped principally at the base of the brain around the blood-vessels. They are most easily recognized by stripping off the pia and holding it obliquely against the light, or floating it in clear water. They are also seen, isolated or in groups, on the dura in the region of the middle meningeal artery and its branches. The choroid plexus is likewise generally filled with tubercular material. The lateral ventricles contain large quantities of a serous, cloudy, sometimes bloody fluid. There may be considerable internal hydrocephalus. The cranial nerves or their sheaths are reddened, covered with exudate, and, at times, swollen. The cerebral substance is markedly injected here and there, adherent to the cortex, and in a condition of red softening at many places. The tubercle eruption and local caseation may also be present at some parts of the cortex, and even extend deeply into the cerebral substance. A slight diffuse or disseminated *encephalitis* of the superficial layers of the cortex is probably always present. Small hemorrhagic foci are occasionally found. Foci of softening may also occur deep in the basal ganglia and cerebral peduncles, due probably to a tubercular arteritis with obliteration (rarely to compression of the vessels by the exudate). The spinal cord may also be invaded.

Symptomatology.—The disease rarely affects strong, healthy individuals, but generally weak, pale children, or adult phthisical persons, who present a poor condition of nourishment. The primary tuberculosis may apparently have run its course, remain latent, or only develop when the cerebral disease arises.

Premonitory symptoms generally precede its occurrence, especially in children who had previously been considered well. They become depressed and peevish, have no pleasure in play, sit with hanging head, brood, and are apathetic at times, at other times irritable, grumbling, and somewhat stuporous. Sleep becomes restless, disturbed by mild delirium, or *insomnia* comes on. *Headache* soon appears, at first periodical and mild, later severe and persistent, even if it exacerbates. The children sometimes complain less of the head than of pain in the chest and abdomen. A motiveless *vomiting* may even occur in this prodromal stage; also a transient increase of the body temperature. Occasionally it is the loss of appetite, constipation, and emaciation which the family notice, and which may precede the outbreak of the disease by some time.

After these symptoms have occurred for one or more weeks, rarely months, the symptoms of *meningeal irritation* become more marked. The transition is generally gradual; at times, however, the *headache, delirium, stupor*, and *convulsions*—the signs of a severe cerebral disease—come on very suddenly. The delirium may be mild or quiet, or severe and combined with marked muscular restlessness. Children who are at this early stage bedridden lie in a restless half-sleep, talk to themselves, make grimaces, cry aloud at times, toss about, or seek to get out of bed. Adults may occasionally still walk around at this stage; they appear mentally abstracted, in a condition of dreamy, excitable confusion; more often, however, the delirium is here also an agitated one; it may simulate that of alcoholism to the slightest detail.

The delirium soon passes into *somnolence*, or these two conditions exist from the first, alternating or combining with each other.

As soon as the sensorium clears up, the patient complains of headache, sometimes also of *vertigo* and of sensitiveness to light and sound. The head pain often breaks through the delirium and somnolence, and betrays itself by the patient groaning and clutching at his head or holding it between his hands. Children often awake from sleep or from the stupor with a piercing shriek (*hydrocephalic cry*). They grind their teeth—though this frequently occurs with normal children—and bore their heads into the pillows. With the onset of the cerebral disease, the vomiting becomes more frequent, obstipation comes on, and the urine, which may be retained or voluntarily discharged, is scanty and contains occasionally some albumin. From the beginning, or later, *motor symptoms* of *irritation* of various kinds appear. The muscular rigidity extends to the muscles of the back, trunk, and extremities; local or general rigidity or rigid contracture is present. A transient tonic muscular contraction of the facial muscles, and those of one arm or one side of the body, also occurs. The maxillary muscles may also be tetanically contracted. The knee-reflexes are exaggerated; they may, however, apparently be lacking, with marked contracture of the legs. Westphal's sign is observed only in later stages; an inequality in the tendon reflexes is not rarely observed. *Transitory muscular twitchings* are noticeable early, now at this, now at that place, particularly, however, in the facial region. The Kernig symptom—rigidity of the knee with flexed hip-joint—is generally present. *General or partial convulsions* occur in this stage. The disease may even commence with an *epileptic attack*. The twitchings often involve one side of the body, resembling the picture of cortical epilepsy, and, following upon such a seizure, indefinite, transient twitchings may repeat themselves for hours. A *tetanic contraction* of the muscles may occur for a short time and recur continually.

The *temperature* is almost always increased, but on an average remains between 100° and 102° F., to undergo at some time a transient rise far above this, or to fall much below the normal temperature. This *irregularity* and fickleness, even in the course of a day, is somewhat characteristic. The pulse is, as a rule, retarded, often arrhythmical, and shows particularly rapid changes in frequency and condition ; only in the last stage does it become very frequent and small. Respiration is moderately accelerated ; severe disturbances generally occur only near the end. The skin is dry, and trophic disturbances generally occur later in the course of the disease, particularly decubitus on typical or uncommon places, for example, on the ear.

The symptoms due to an affection of the cranial nerves are of great diagnostic importance. On account of the basal extension of the process, they almost always occur, though generally only at the end of the first or in the course of the second week. The nerves supplying the ocular nerves are particularly involved, producing a unilateral or bilateral ptosis, pupillary contraction or dilatation, unequal pupils, absent reaction, etc. The eyeballs are turned lateralward, or upward or downward, or they roll here and there from one extreme position to another ; *nystagmus-like twitchings* and paralytic strabismus also are among the symptoms. An ophthalmoscopic examination reveals an *optic neuritis* or choked disk in many cases. Tubercles are sometimes noticed in the choroid, though not so often as in miliary tuberculosis. Of the other nerves, the *facial* is the most often attacked. Paresis and even paralysis may occur in addition to the twitchings and contractures already spoken of. Contracture and paralysis may manifest themselves together. Sometimes the facial paresis is first noticed from the partial closure of the eyelids.

The truncal muscles are attacked by paralysis at the same time.

Monoplegia of an arm, or complete *hemiplegia*, appears. It may extend from one side to the other. These paralytic conditions are sometimes only transient ; occur after a spasm, to soon disappear ; they often, however, persist, and in some cases hemiplegia is a prominent initial symptom. *Aphasia* is not uncommon, and is often an early symptom.[1]

Disorders of *sensibility* are rarely noticed. The only one which appears, as a rule, to any degree, is an *hyperesthesia* of the skin and muscles, particularly in the initial stadium of the disease. Attempts at passive movements, also, generally cause pain.

In the second, but particularly in the beginning of the third week, the *coma* deepens more and more; the patient cannot be awakened, and lies unconscious. The paralytic symptoms increase in intensity and extend further. Vomiting ceases. The pupils become dilated and reactionless. The cheeks are sunken and the emaciation is considerable. Even in this stage, *remissions* occur, which induce the uninitiated to expect a recovery. But the relapse soon follows. Finally, the rigidity disappears and gives place to a general relaxation; the patient can no longer swallow; respiration becomes irregular, assumes a Cheyne-Stokes type, etc. The pulse, until then retarded, becomes suddenly or gradually very frequent; and, after a rapid rise in temperature to 106° F. or more, or a considerable drop, as low as 73° F., *death* comes on.

---

[1] The focal symptoms may be due to inflammatory edema, to pressure of an exudate, to local meningo-encephalitis, and finally to a softening caused by an arteritis obliterans. (Friedlander, Cornil, Chantemesse, Zappert.)

Course and Prognosis.—The disease may take a violent course and end in death within a few days. It generally lasts two to three weeks; counting the prodromal stage, several months. It generally takes a steady, progressive course, though remissions often occur. The distinction between an irritative and a paralytic stage can rarely be sharply made. The symptoms of excitation are always prominent in the first week, while later the phenomena of depression and paralysis are in the ascendency.

Some cases are thoroughly atypical in course and prognosis. This is particularly true of tubercular meningitis in adults (Boix, Jaccoud). It may even remain latent, or be overshadowed by the phenomena of the primary disease (Wunderlich, Fraenkel).

The increase in temperature may be completely absent. That this disease may resemble delirium tremens has already been stated. It may commence with the symptoms of a focal disease,—monoplegia, hemiplegia, aphasia, cortical epilepsy,—and continue uncomplicated, with only these symptoms, for some time (Rendu, Chantemesse, Weintraud), though in such cases it is probably originally a local tubercular meningo-encephalitis (see below), which only later becomes generalized. Finally, though rarely, chronic cases of tubercular meningitis occur in which even the intermissions seem to be of longer duration.

The prognosis is bad. Most authors doubt that a recovery ever occurs. It cannot be doubted, from the observations of Dujardin-Beaumetz, Rilliet-Barthez, Schwalbe, Politzer, Janssen, and particularly from a case reported by Freyhan, in which tubercle bacilli were found in the fluid obtained by spinal puncture, that recovery may ensue.

We sometimes see mild meningeal symptoms come on in the course of tuberculosis, which soon disappear. It is difficult to determine whether these are cases of beginning tubercular meningitis aborted in the embryonal stage, or whether the *tubercular toxine has caused temporary cerebral symptoms.*

The preceding chapter should be consulted concerning the **differential diagnosis**. We have also considered there the diagnostic importance of lumbar puncture, a positive result thereby (discovery of tubercle bacilli in the liquid) being of great value. The fluid is generally clear; it may, however, be also cloudy and purulent, rarely bloody and particularly opalescent. The pressure is very high and the liquor coagulates easily. Absence of tubercle bacilli in no way, however, indicates that there is no tubercular meningitis present.

It should be easily distinguished from *tetanus*. In a case in which the disease commenced with trismus, tubercular foci were found at the foot of both central convolutions. Confusion with typhoid and other infectious diseases is possible, particularly in children. In the prodromal stage this severe cerebral disease is often not recognized; a harmless gastro-intestinal catarrh, anemia, etc., is thought of. It is just as wrong—and the error is often made—to think of a commencing meningitis at every complaint of a tuberculous or scrofulous child, when the appearance is haggard and the child is depressed. Scrofulous children of tubercular parents are often *nervous;* and, through this, anomalies of disposition, loss of appetite, emaciation, and acceleration of the pulse occur, without being of much importance.

Tubercular meningitis is not always easily diagnosed from *miliary tuberculosis*. In the latter, however, the *pulmonary* symptoms are very prominent, and a slight increase in respiration and pulse is generally observed from the beginning. Miliary tuberculosis may also, however, be combined with tubercular meningitis.

The tubercular form differs from the other meningitides in its longer prodromal stage, the less violent course, the, on the whole, inconsiderable rise of temperature, etc. In childhood this form should always first be thought of, though serous exudates are also relatively frequent in childhood. Very important is the detection of choroid tubercles.

In commencing caries of the petrous bone, a purulent as well as a tubercular meningitis may be present; in children, it combines more often with the latter (Henoch).

Finally, those forms of *local tubercular meningo-encephalitis* must be mentioned in which — generally on the convexity — a circumscribed meningeal tuberculosis with caseous tubercles extends to the cortex and forms the only alteration. The cortical substance in the neighborhood of the lesion is generally softened.

In such cases the clinical picture does not represent that of tubercular meningitis, but of a cortical disease, and mostly that of solitary tubercle. I found, in a man who suffered with hemichorea and psychic disorders, in addition to a solitary tubercle in the thalamus, a tubercular meningo-encephalitis over the right frontal lobe. In another case there developed, with headache and right-sided convulsions, an aphasia, which persisted while the accompanying symptoms disappeared. The autopsy, made years after, revealed a local tubercular meningo-encephalitis over the speech centre.

Treatment.—The chapter on purulent meningitis is also to be consulted for the treatment. The hopes which some had in iodoform have not been realized. The prophylaxis of tubercular meningitis is similar to that for tuberculosis in general, and further consists in combating it or every tubercular lesion.

Surgical removal of all foci before the virus has been carried to the brain is proper, if practicable. Children with a tubercular predisposition should be guarded against mental strain. Careful nursing and a sojourn in fresh, good air are also prophylactic. In one case recovery occurred under the administration of enormous doses of potassium iodide (from one hundred and twenty to six hundred grains a day).

Surgical treatment has till now not done much good. The opening of the cerebral subarachnoid spaces from behind, with after-drainage, has been recommended and done (Morton, Parkin). Ventricular tapping has been used as a palliative remedy (v. Bergmann, Keen, and others).

The least harmful of these methods is lumbar puncture, which has been followed by recovery in a number of cases. It was also employed in Freyhan's case, though the author was not inclined to refer the recovery to it.

The individual symptoms must be treated symptomatically.

CHRONIC MENINGITIS.

Excluding the syphilitic forms, discussed in the section on cerebral syphilis, chronic meningitis is a rare affection. Chronic meningitis occurs in cases of *chronic alcoholism*. It is found especially on the convexity of the brain, and is characterized by a slight cloudiness and thickening of the soft cerebral membranes. The same alterations are observed in the diffuse diseases of the cerebral cortex, especially in dementia paralytica, senile dementia, and hereditary chorea.

We know nothing definite concerning the symptomatology, as the disease is generally an unexpected post-mortem discovery, or its symptoms are overshadowed by those which are induced by the cerebral disease. *Chronic ossifying meningitis*, which is often found in individuals mentally diseased, in epileptics, and also sometimes in persons who have suffered for years with a persistent headache, is likewise hardly of any clinical interest. It may lead to the formation of bony layers which surround the cortex like a cap.

Chronic basal meningitis is better understood. In most cases it is of syphilitic origin. It has several times occurred as the residuum of an acute cerebrospinal meningitis which has run its course. Several cases have been observed, however, which indicate the occurrence of a *simple primary chronic basal meningitis*. It produces cloudiness, thickening, and adhesions between the membranes and the cerebral cortex;

the cranial nerves also become involved, especially the *optic* nerves. It is of considerable importance from the fact that the communications between the ventricles and the subarachnoid spaces become displaced or dammed up, causing internal hydrocephalus.

Some authors have gone so far as to aver that all cases of acquired (idiopathic) hydrocephalus are due to such a basilar meningitis. This is, however, not correct. (See chapter on Hydrocephalus.) On account of the small number of cases that have come to an autopsy, it is not possible to construct a characteristic picture for this simple basilar meningitis. As a rule, persistent headache, at times exacerbating, occurred; also vomiting and, more rarely, vertigo and general spasms. A slight rise in temperature has also been noticed. *Optic neuritis*, ending in *atrophy* and at times paralysis of the ocular muscles, has been observed. It takes a chronic course; may cease from progressing; or hydrocephalus may cause death.

In infants a chronic meningitis entirely or mostly in the posterior cranial fossa occurs, and is probably of a syphilitic nature. The meninges are thickened, and adhesions between them and the cerebellum, medulla oblongata, etc., are present. Nuchal rigidity, vomiting, general spasms of an epileptic and tetanic nature, and rigidity of the muscles of the trunk and extremities are symptoms. Blindness is also often present, the result of a secondary hydrocephalus, which presses against the optic chiasm on the floor of the third ventricle. In one of my cases total amaurosis with intact pupillary reflexes, and without any ophthalmoscopic alteration, was present, so that I suspected an extension of the process to the occipital lobes. Paralysis of the ocular muscles and nystagmus have likewise been observed. Finally, the cranial nerves arising from the posterior cranial fossa may be attacked, and paralytic symptoms corresponding to this involvement be found. The prognosis is grave; the children generally die from hydrocephalus. The meningitis may, however, cease progressing and recovery take place. An antisyphilitic treatment is always indicated in cases of this nature.

CIRCULATORY DISORDERS OF THE BRAIN.

Our knowledge of the circulatory disturbances in the brain and the phenomena produced thereby is deficient, as pathologic anatomy has rendered but little if any assistance. The amount of blood found in the brain post mortem does not give an absolute indication of the quantity which the organ contained during life, as this is to a large extent dependent upon other factors,—the manner of death, the position of the corpse, etc. Our observations are based upon cerebral phenomena which occur in persons suffering from general anemia or plethora, as well as upon symptoms produced by a sudden loss of blood or sudden interruption of the blood supply to the head. The phenomena produced experimentally—by ligating the afferent vessels to the brain—are of doubtful value in the determination of the circulatory disorders in the human brain. Kussmaul and Tenner have produced general spasms and loss of consciousness by ligating the carotid and vertebral in animals. Unconsciousness has also been observed in man after compression of both carotids.

CEREBRAL ANEMIA.

Cerebral anemia develops acutely after a sudden and pronounced loss of blood or after the withdrawal of large quantities of blood to other regions, as, for example, in paracentesis of the abdomen and sudden evacuation of an extensive ascites, precipitate births, and the use of Junod's cupping boot; also by inhibition of the blood supply to the brain from acute cardiac weakness or spasm of the cerebral arteries.

Pallor of the face and mucous membranes generally occurs at the same time as the cerebral anemia, though not always.

If the *cerebral anemia* occurs acutely, the following *symptoms* are produced: the affected individual is attacked with *buzzing in the ears*, *nausea*, at times *vomiting;* everything appears dark before him; he feels the floor giving way beneath him; his senses are less acute than usual, *apathy* and a desire for *sleep* set in; the *pupils* are *contracted*, and finally consciousness may be entirely lost. When a large quantity of blood has been lost the *unconsciousness* is complete, the reflexes are annulled, the pupils dilated, etc. Generalized spasms may also occur. Naunyn was able to induce, by compression of the carotids in individuals affected with arteriosclerosis, the following symptoms: unconsciousness, spasms, dilatation of the pupils, and retardation of the pulse. He ascribed these symptoms to cerebral anemia due to defective blood-pressure within the circle of Willis. Milder types correspond to the picture of *syncope.* The patient feels badly; his mind is not so clear as formerly; yawning occurs, and tinnitus aurium and flickering before the eyes manifest themselves. The skin becomes pale and cold, and is covered at times by a cold sweat. Consciousness becomes lost, but usually only partially, so that strong sensory stimulations are still felt. The pulse is small, generally somewhat accelerated, also perhaps intermittent; respiration, retarded or accelerated, gasping and irregular. Convulsions do not occur. The attack has, as a rule, a duration of some minutes; it may, however, last an hour. Consciousness repeatedly returns upon the patient reclining, to disappear as soon as he sits up again.

The cause of these seizures of syncope is the sudden stoppage of the blood supply to the brain. This may be produced by a general spasm of the cerebral arteries or be due to defective cardiac action; the latter factor is probably commonly in play. The attacks may be produced by terror, anxiety, painful sensory impressions (seeing blood), severe pains, etc. The individuals attacked are almost always of a nervous disposition; general anemia increases the tendency to syncope.

Chronic anemia of the brain in chlorosis, pernicious anemia, after repeated loss of blood, etc., produces *heaviness of the head, drowsiness,* *apathy*, tendency towards *vertigo* and *syncope*, *tinnitus aurium*, *weakness* of the *memory*, *insomnia*, rarely hallucinations. These symptoms increase upon assuming an erect position; the patient feels better when reclining. One of my patients, who was very anemic as a result of former blood-losses, became affected with the following symptoms, only, however, in the morning, after arising: first, a mist before the eyes, then severe vertigo, and finally vomiting. The attack lasted from an hour to two hours; it was shortened, however, when the patient lay down.

The delirium of *inanition* can, in part, be referred to anemia of the brain, though chiefly due to defective nourishment. Persistent diarrhea may, particularly in children, produce symptoms of cerebral anemia. The most severe forms are those described by Marshall Hall as *hydrocephaloid*. The cerebral symptoms occurring at times in chlorosis, which may even simulate the picture of a tumor, are probably mostly due to complications, as sinus thrombosis, serous meningitis (?), etc.

The prognosis of cerebral anemia is good in the milder forms; attacks of syncope are hardly dangerous to life. Even where it can be determined that an habitual disposition to syncope is present, recovery should be looked for. When considerable blood has been lost, coma may occur and take a fatal course. The onset of general convulsions is always a grave sign, as are also dilatation of the pupils and loss of the light-reflex. If they contract again, and if the reflex returns, the prognosis as to life is good. Visual disorders and optic atrophy may, however, be a permanent result of the loss of blood.

Treatment.—In acute cerebral anemia a *horizontal position*, perhaps with the head hanging down, is imperatively demanded. The flow of blood to the brain may also be increased by binding the extremities firmly from the distal ends towards the body. If defective cardiac action or syncope be present, *excitants* are in place,—wine, cognac, ether, camphor. The skin should also be stimulated by sprinkling with cold water, applying mustard plasters, faradization, etc. *Amyl nitrite* may be tried in persistent cases. A few drops should be placed upon a handkerchief and held under the patient's nose. In severe cases of cerebral anemia, *artificial respiration* and even transfusion may be necessary.

The treatment of chronic cerebral anemia is similar to that of general anemia.

HYPEREMIA OF THE BRAIN.

It may be *active* or *passive*. It is doubtful if there ever occurs a persistent overfilling of the brain with arterial blood; just as doubtful as is the occurrence of plethora itself. There are, however, individuals of short stature, short neck, and with a continuous red face (or after every meal and after physical exertion), in whom the phenomena we refer to hyperemia of the brain are present constantly or appear upon the slightest cause. The view that continued mental exertion produces a chronic arterial hyperemia of the brain is not based upon accurate data.

More exact is our knowledge of transient increase of the arterial blood supply to the brain. That this is the condition in so-called *congestion* is hardly to be doubted.

Some persons often suffer from attacks without any known reason, or after excitement, the ingestion of spirituous liquors, large meals, etc., attacks which are described as a rush of blood to the head, a fluxion, or active congestion. They become suddenly hot; the heat rises to the face and head; they feel a throbbing in the temples, neck, and also in the head; they see a mist or flickering before the eyes; headache and vertigo come on, also a dazed feeling; after some minutes or a half-hour the attack in mild cases is over. During the seizure the face and neck become intensely red; the conjunctivæ may also become injected; the skin of the head feels hot, especially around the ears; the pulse is retarded or accelerated, full and tense; the pupils may be contracted; and the carotid and temporal arteries throb violently.

In severer cases a deeper disturbance of consciousness occurs, either complete unconsciousness or a dazed condition, perhaps combined with a sort of maniacal excitement. Fever has also been observed. In children and youthful individuals conditions of this kind have been described as active hyperemia of the brain, in which the phenomena reached such an intensity as to simulate the picture of a most acute meningitis, yet disappeared in a few hours or in the course of a day. A paralytic form of arterial hyperemia has also been described, an apoplectic attack with transient paralysis (hemiplegia), or even with a fatal termination, the necropsy revealing nothing. It has not, however, been proven that a simple hyperemia will evoke paralytic conditions. That amyl nitrite will produce temporary aphasia is, however, worthy of notice. Generalized and even unilateral convulsions may be the result of an active congestion of the brain.

The rush of blood to the head is probably due to an inherited or acquired instability or "lability" of the nervi vasorum system, which induces, from relatively slight causes, *dilatation of the cerebral vessels*, especially of the capillaries, accompanied by an increased cardiac action. It occasionally accompanies neurasthenia and hysteria. Masturbation plays a prominent part in the production of this condition.

Another cause of active cerebral hyperemia is decreased blood supply to other parts of the body. Sudden contraction of the peripheral vessels in a cold bath may cause fluxion to the brain. An analogous action is said to be possible from repression of habitual hemorrhages (menses, hemorrhoids). Certain poisons increase the flow of blood to the brain by dilating the cerebral vessels; to these belong alcohol, nitroglycerin, amyl nitrite, etc.

Some authors refer the phenomena of *insolation* to cerebral hyperemia.

Venous or *passive hyperemia* is generally a chronic condition, produced by compression of the vessels carrying the venous blood from the brain (the jugular) by tumors of the neck, struma, etc., and of the superior vena cava by mediastinal tumors etc., also by uncompensated heart disease, by pulmonary emphysema, etc. Coughing, sneezing, straining, bending, tight collars, etc., may produce a slight venous hyperemia of the brain or increase an existing one.

The symptoms are *sleepiness or sleeplessness,* a feeling of weight in the head, which is increased on coughing, vertigo, apathy, somnolence, and slight confusion of the mind.

These symptoms are generally, though not always, more marked in a recumbent position than in an erect one; the conditions of fear and mental confusion, which may occur at every attempt to sleep, are particularly bothersome. In severe cases, sopor and coma may come on, perhaps epileptic attacks.

The phenomena of passive congestion are generally also recognizable upon the face and mucous membranes.

The prognosis of simple congestion is, on the whole, favorable. When it is due to some organic disease of the vessels, heart, etc., there is some danger.

The prognosis of venous hyperemia conforms to that of the cause; it is almost always bad.

Treatment.—In order to guard against a rush of blood to the head excesses of all kinds are to be avoided; also the use of strong coffee and tea; a very small quantity of intoxicating liquors may be allowed, and then only a good beer or a very mild wine (even Rhine wine is too heavy). A rubbing down with cold water and a warm half-bath are the hydropathic methods to be used in counteracting a tendency to congestion.

In the attack itself, the patient should assume a sitting or recumbent position, with head elevated, the neck bared, and the room cool and well aired. Cold compresses upon the head, sinapisms to the back of the neck, breast, and calves of the legs are advisable in mild attacks. Faradization of the skin of the trunk and particularly the feet may do much good. If the seizure is severe or long drawn out, leeches to the temples, cupping-glasses, or even venesection may be used in plethoric persons; active purgation (calomel, irritating clysmata, etc.), hot foot-baths, etc., being also in place. It is improbable that ergot is able to counteract cerebral hyperemia.

Venous congestion can only be combated by allaying the cause. Extirpation of a tumor or struma, regulation of the heart's action with digitalis, etc., may be demanded.

Symptomatic treatment consists in purgation, and in relieving the headache and insomnia by narcotics. The bromides may be recommended, but the use of opiates and chloral hydrate is not without danger.

DISEASES OF THE BRAIN-SUBSTANCE.

CEREBRAL HEMORRHAGE.

Hemorrhage into the cerebral substance forms one of the most frequent diseases of this organ. It may come on at any age. Most cases, however, occur in old age; it is rare before the fortieth year of life. Males are more often affected than females.

The hemorrhage is almost always a result of *vascular disease*. The most frequent disease of the cerebral arterial system is *atheroma*. While this affects principally the larger vessels, the hemorrhage comes mostly from laceration of the arterioles coming from the arteries of the circle of Willis, particularly the intracerebral branches of the middle cerebral artery. *Miliary aneurisms* seem to favor these branches (Charcot and Bouchard). They may occur in conjunction with arteriosclerosis, but are not always combined with it. Miliary aneurisms rarely develop before the fortieth year; after this age, with increasing frequency. Generally rupture of a miliary aneurism may be detected as a cause of the cerebral hemorrhage, though this should not be considered a condition *sine qua non*. The frequency of this disease of the vessel wall has been overvalued, as, for instance, simple excavation of the lymph sheath from an effusion of blood has wrongly been called an aneurism (Egger, Stein).

Other diseases of the cerebral vessels may produce rupture and hemorrhage. Small hemorrhages may perhaps arise from diapedesis. Hemorrhages due to laceration of aneurisms of the larger vessels are mostly found in the meninges, especially at the base of the brain; they will not be considered here.

Cerebral hemorrhage may also be due to laceration of veins and of the venous sinuses.

The causes of disease of the cerebral arteries are manifold. *Old age* is the most important. Then follow *intoxication* and *infection*. Alcoholism and lead intoxication not rarely, syphilis quite often, figure as causes of the injury to the cerebral arteries. These conditions, therefore, play a part in the etiology of cerebral hemorrhage, although syphilitic arteritis more often occurs in softening than in hemorrhage. The acute infectious diseases also favor the onset of cerebral hemorrhage, though its occurrence from them has not been frequently noticed. It is assumed that they may cause arteriosclerosis and other diseases of the vessel walls. In the general diseases which produce a *hemorrhagic diathesis*, as purpura, pernicious anemia, and leukemia, the brain is not rarely the seat of—generally multiple—hemorrhages. *Gout* and *nephritis*, especially the atrophic kidney, may also increase the predisposition to cerebral hemorrhage. It has likewise been often found in the eclampsia of pregnancy.

Heredity is not without importance as a causative factor. A tendency to "strokes" may be inherited through generations. Hemorrhage due to obstruction of a blood-vessel is of minor importance.

If the cerebral vessels are diseased, hemorrhage may occur without any other factor coming into play. Its occurrence is, however, favored by all movements which increase the blood-pressure. *Hypertrophy of the left venrticle*, especially when not compensated, is always dangerous to the brain. It is improbable that healthy vessels rupture under such conditions. The relationship existing between cerebral hemorrhage and kidney diseases is probably in part due to this circumstance; on the other hand, granular atrophy of the kidney is often combined with an arteriosclerosis of the vascular system.

A temporary increase in blood-pressure is often the exciting cause of the cerebral hemorrhage. In this way *severe muscular* exertion may superinduce it; also forced expiratory movements, as in coughing, sneezing, straining at stool and in parturition, etc. In the rare cases of cerebral hemorrhage in children, as noticed in the course of whooping-cough, this movement seems to be the cause. Sudden and intense emotion, as terror and anger, is an exciting cause. It is improbable that such a cause can produce rupture of a normal cerebral vessel, but its possibility must be acknowledged. I treated a healthy sixteen-year-old girl, who, upon being informed of a death, sank to the floor unconscious, and awoke with the symptoms of cerebral hemorrhage.

A cold bath, coitus, and alcoholic excesses are also exciting causes. A young, strong, and till then healthy officer, not given to the drinking of alcohol at other times, and without the signs of any vascular disease, had a stroke soon after an excessive drinking bout (Kneiperei), and was ever afterwards afflicted with a hemiplegia.

In contrast, to some extent, to these experiences, is the fact that cerebral hemorrhage at times occurs in *sleep*. Gowers believes that perhaps the difficulty entailed by the blood in flowing from the brain in a recumbent position is the cause of this.

Cranial injuries excite particularly meningeal bleeding; they may, however, also cause hemorrhages within the cerebral substance. Days and weeks after a cranial contusion, hemorrhages may still occur in the neighborhood of the aqueduct of Sylvius and the fourth ventricle, generally preceded, however, by a local softening of the tissues.

Location of the Hemorrhage.—Every part of the brain may be the seat of hemorrhage, though not with equal frequency. Its favorite locations are the central ganglia, the corpus striatum, the optic thalamus, and the neighboring part of the internal and external capsules; then follow the centrum semiovale, the cortex, and the pons. Cerebellar hemorrhage is rare; still more so is that of the corpora quadrigemina and the medulla oblongata. It should be remembered that the medulla oblongata embraces a relatively small extent.

In accordance with this arrangement of the hemorrhage in the different cerebral provinces is the fact that miliary aneurisms are most often found in the arteries of the central ganglia, most rarely in those of the medulla oblongata. The lenticulo-striate and the lenticulo-optic arteries are the ones in which aneurisms most often develop (in seventy-five per cent. of all cases, according to Durand-Fardel).

Those arteries which arise at a right angle from the arterial stem, and which are of rather large calibre in addition to being *terminal arteries*, are under a relatively high pressure, in contrast to the vessels of the cortex, which branch a great deal, ending in a net-work, and at least have small anastomoses.

Pathologic Anatomy.—Miliary aneurisms, which can be seen with the naked eye, have a diameter between one-tenth and one millimetre. They produce an ampulla-like dilatation or excavation of the blood-vessel, though we should only speak of aneurisms when the arterial wall is diseased. The process commences in the muscular coat, which disintegrates and atrophies (Roth, Arndt, Loewenfeld). Finally, the entire vessel wall is involved in the degeneration and excavation.

The size of the coagulum varies. It may have the circumference of a man's fist, and upon breaking through into the ventricle may attain a still larger size. The large foci are found in the region of the central ganglia and centrum semiovale; while those in the cortex, cerebellum, and pons are generally much smaller. Multiple clots in general hemorrhagic diatheses and acute infectious diseases are usually small. In the clot the blood is for the most part mixed with broken-down cerebral substance, the neighboring brain substance being also ragged and filled with small clots. The appearance of the clot varies according to its age: it may have the color and characteristics of ordinary fresh blood, or it may have been converted into a brown, brownish-black, or, later, a yellow mass It retains a hard consistency for about four weeks; after about five weeks the clot shows a yellow color (Monakow), and after two to three months becomes ochre colored. Upon microscopic examination we find in the clot fat droplets, granular and crystalline *pigment* (hemoglobin, hemosiderin, hematoidin). Some idea of the age of the exudate can be obtained from the decoloration, swelling, atrophy of the red blood-corpuscles and their inclusion in contractile cells, and from the nature of the pigment. Cells inclosing blood-corpuscles appear only on the third day. Upon the eighteenth day Dürck found the first evidence of, and after the sixtieth day exclusively, free pigment.

A longer time is necessary for the formation of an *apoplectic cyst;* it is assumed that it can only develop, at the earliest, in from three to four weeks. The neighboring brain-substance forms, then, from proliferation of the glia, and probably also from connective-tissue formation, a kind of capsule around the clot, the contents of which gradually become absorbed, and a space filled with serous fluid remains. Complete cicatrization occurs rarely (this is more often observed after softening).

If the bleeding is extensive, and death has occurred early, the convolutions of the same side of the hemisphere are flattened, the sulci are somewhat smoothed over, and the falx cerebri is pushed towards the other side.

Symptoms.—Though the phenomena of cerebral hemorrhage are to a certain extent dependent upon the seat and extent of the hemorrhage, we are nevertheless able, in almost every case, to distinguish two groups of symptoms, the first temporary, the second constant. The immediate result of the cerebral bleeding is the *apoplectic insult* (of the Germans), the "stroke," as it is popularly called; the lasting result of this cerebral lesion is paralysis, and, as a rule, unilateral paralysis (hemiplegia).

The Apoplectic Attack.—The patient may suddenly, without any prodromes, fall unconscious to the floor (foudroyant apoplexy). More often, however, certain disturbances of the general health precede the attack and warn the patient at least some minutes before the onset of unconsciousness. These prodromes consist in a feeling of vertigo, of blood rushing to the head, of a heaviness of the head, of "darkness before the eyes," of cardiac palpitation, or of paresthesia of one side of the body, mental confusion, and speech disturbance. Retinal hemorrhages and bleeding from the nose may also be prodromes. These disorders may occur temporarily days or weeks before the attack, though this is not the rule. The individual is generally in good health when the attack comes on.

The patient lies in a *comatose* condition and on superficial examination resembles a sleeping person. The voluntary movements and sensations are abolished; he cannot be awakened from his sleep; his face is generally red and bloated. The pupils do not react to light, are of normal width or, more rarely, abnormally wide (contracted only in pontile hemorrhage); the conjunctival and corneal reflexes are missing. The musculature is flaccid; the passively raised extremities fall lifeless to his side; even the deep reflexes may be absent in this stage, and the peripheral reflexes are always lacking. The pulse is generally full and strong, of normal frequency, retarded, or, rarely, accelerated. The respiration is generally retarded and deepened, often stertorous, the inspiratory rush of air moving the flaccid palatine velum and vocal cords. Cheyne-Stokes breathing occurs more rarely. The cheeks are puffed out excessively during expiration. Fluids brought into the mouth are not swallowed. The urine and feces are passed involuntarily; the quantity of urine may be increased, contains albumin, at times also sugar (though rarely longer than the first twelve to twenty hours after the attack).

Generalized or unilateral convulsions rarely occur during a seizure. The temperature sinks in the first few hours from one to two degrees, then rises to normal or somewhat above it. A marked and permanent decrease in temperature is an ominous sign, just as much as is a rapid and excessive increase.

Such an attack has a duration of some hours (on an average, one to four) or from one to two days.

The chief cause of the coma is probably the circulatory disturbance (anemia) in the cerebral cortex, produced by the pressure of the blood extravasation and other factors. Molecular concussion, transmitted from the location of the hemorrhage more or less through the whole brain, may also cause an inhibition of cortical functions (also applicable to emboli). Monakow believes that a contraction of the cortical arteries produced by reflex action plays a part in the genesis of the coma.

Deep coma is generally replaced after some hours by somnolence, the corneal and other reflexes return, the patient can swallow, and executes transient active movements, betraying by them the side on which the paralysis is located.

The stage of the insult, or seizure, passes, as a rule, in from twenty-four to forty-eight hours into that of the *reaction* (corresponding to the reactive occurrences in the blood-clot and its neighborhood). The pulse becomes more frequent, the temperature increases to a normal height or higher, the skin becomes covered with perspiration; headache, slight delirium, and sensory and motor symptoms of excitation may come on. These disturbances are mostly only of short duration.

The apoplectic attack does not always induce a deeply comatose condition. At times only simple loss of consciousness or merely slight mental confusion is present, and the phenomena accompanying coma described above are absent. Even a vertiginous sensation of slight duration or a transient stupor may take the place of the apoplectic seizure. Finally, the paralytic symptoms may come on with intact sensorium and without any apoplectic attack.

These differences greatly depend upon the extent of the hemorrhage, the rapidity with which it comes on, and its location. When the hemorrhage as usual occurs quickly, and is larger than the size of a cherry or a hazelnut, it is characterized by a greater or less degree of unconsciousness,—*i.e.*, by the apoplectic attack. Hemorrhages which involve the cerebellum or pons less often entail unconsciousness than do cerebral hemorrhages.

Vomiting is one of the *inconstant* symptoms accompanying the stroke, and is seen most frequently in cerebellar hemorrhages. So is the unilateral deviation of the head and eyes (in cerebral hemorrhages towards the side of the lesion). General and unilateral *convulsions* and delirium rarely usher in or accompany the apoplectic attack.

If death does not occur during the attack and the patient gradually recovers consciousness, focal symptoms become evident as a result of the apoplexy, the most frequent being a *hemiplegia*. The paralyzed side may even be discovered before the movements can be executed. Careful observation reveals an asymmetry of the face, in that the oral angle stands lower on the paralyzed side; the cheek is puffed out more, the nasolabial fold is less marked or completely blotted out, and the saliva drools from the angle of the mouth on this side.

If the extremities are passively raised, they fall lifeless on both sides during complete coma; but as soon as the sopor lessens it is noticed that the muscle-tonus of one side is still, at least partly, present, while on the other side the limb is completely flaccid. Motor phenomena of excitation must not be confused with active movements.

The condition of the reflexes is also of service, the *cremasteric* and *abdominal* reflexes, for instance, being abolished on the paralyzed side (Rosenbach, Jastrowitz).

The condition of the respiratory muscles may at times reveal the side paralyzed during the comatose period, in that the corresponding half of the breast and the diaphragm do not respond quickly to respiration (Nothnagel, Grawitz, Ortner), and that the diaphragm moves less readily.

If conjugate deviation of the eyes is present, the seat of the lesion is almost always upon the side towards which head and eyes are turned.

Active movements are gradually resumed in the limbs of one side of the body. The legs or arms should be manipulated and brought into an uncomfortable position, when it can easily be noticed how the will can bring them from this position on the normal side, in contrast to those of the paralyzed extremities.

If the apoplectic attack is missing, paralysis is the first symptom noticed. Any object which the patient grasps falls from his hand; his legs bend beneath him, and he is compelled to sit down or he collapses.

Hemiplegia is the typical result of an apoplectic attack,—*i.e.*, paralysis of the arm, leg, facial and hypoglossal of one side of the body. (See page 437.) The deglutitory, laryngeal, and truncal muscles are almost always spared, only the trapezius tends to take part in the paralysis.

Soon after the attack, hypesthesia is noticed upon the paralyzed side of the body. Speech becomes somewhat indistinct and heavy, a slight dysarthria being present, which disappears in some days or leaves behind a slight speech defect.

This is the clinical picture in typical cases where the hemorrhage has occurred in the region of the central ganglia, and the motor conducting tracts in its course through the posterior limb of the internal capsule is injured directly or indirectly.

If it is directly affected by the hemorrhage, and is more or less completely disturbed, the hemiplegia remains, constituting a direct focal symptom.

The clinical picture, however, varies soon afterwards,—sometimes in a few days, at times even after some hours,—an *exaggeration of the deep reflexes* becoming apparent on the paralyzed side so that an ankle-clonus may be elicited.

This increase in the deep reflexes affects generally—though not so markedly—the extremities, particularly the leg, on the *sound* side; a slight decrease of motor strength being also evident in the muscles of the sound side. I have several times found a mandibular reflex as the only expression of this bilateral exaggeration of the reflexes.

After a few weeks, a certain degree of mobility appears in the paralyzed limb, the patient gradually secures control of it so that he can, when reclining, elevate and extend his leg, though slowly and without much strength. Either the arm remains entirely paralyzed or minimal movements are reacquired; as, a slight abduction of the upper arm, a slight flexion of the lower arm, or a few sluggish and feeble movements of the fingers. The paralysis in the extensors of the hand and fingers generally persists; while a certain degree of movement is re-established in the flexors, so that the patient can shut but not open his hand. Of the muscles of the leg, the flexors of the knee and the extensors of the foot and toes are those that are permanently paralyzed (Wernicke, Mann). At any rate, the arm remains more involved than the lower limb. A hemiplegic almost always again acquires the ability to walk, even though only after some months, while the arm remains, as a rule, useless.

Muscular contraction and shortening, *contractures*, have almost always come on, however, in the mean time.

The arm is here again chiefly involved and is fixed in a certain position, to which it always returns when moved: the upper arm is adducted, the lower arm flexed, the hand generally pronated and usually slightly flexed at the same time, the proximal phalanges are somewhat and the middle and distal phalanges more strongly flexed.

If an attempt be made to bring the extremity into another position, the muscular tension must first be overcome, which may be partially or completely possible, but the arm immediately returns to its former position upon removal of the force. If it be desired to bring the finger from a flexed into an extended position, this is generally possible only after the hand is brought into a position of extreme flexion. The deep reflexes are markedly exaggerated; wrist-clonus may occasionally be present.

The deformity produced by the contracture is less marked in the leg, because it is fixed in a position of extension. Exceptions are rare; a flexion-contracture of the legs occurs, at times, when the patient is continually bedridden.

A contracture of the Achilles tendon and the equinus or equinovarus position of the foot produced thereby give a characteristic position to the foot and form a considerable hinderance in walking. The patient supports himself more on the sound limb, draws the paralyzed one after him and circumducts it, the foot describing a half-circle. He, at the same time, lifts the pelvis upon the paralyzed side and draws it in a circle, passing from behind forward, around the resting leg as an axis. He can, at the same time, not lift his toes from the floor.

The paralyzed arm is generally fixed by the sound arm in walking, if there is any tendency to sympathetic or associated movements.

Contracture is rarely observed in the face; in permanent hemiplegia, we occasionally notice that the mouth is drawn to the paralyzed side and that the nasolabial fold is more deeply furrowed here.

In a few cases, the paralysis which remains is a flaccid one, without there being any explanation known for this deviation from the ordinary rule. We have already referred (on page 440) to the motor symptoms of excitation of a different type which at times occur in the previously paralyzed limbs.

The musculature generally retains its normal volume or suffers a slight atrophy with a minimal quantitative decrease of electrical excitability as a result of inactivity (see above).

If the hemorrhage has not directly involved the motor conducting tract, but merely injured it by compression, the hemiplegia is not a lasting one; it either disappears rapidly or within some weeks or months, and has the character of an *indirect focal symptom*. It can easily be understood that between these two forms, the direct and the indirect involvement of a conducting tract, there is no sharp border; *e.g.*, the clot may be in the thalamus or in the lenticular nucleus, and act for the most part by pressure, and yet impair directly some of the motor fibres.

If the hemorrhage extends to the most posterior region of the internal capsule (rupture of the lenticulo-optic artery or of the choroid artery), *hemianesthesia* occurs in addition to the hemiplegia. This is rarely a total one, there generally being only a more or less marked decrease of sensation, for all or only for some qualities. It is either present over the whole side of the body or is marked only in certain parts (especially the extremities).

In rare cases the hemorrhage confines itself to the region of the sensory conducting tract, a complete hemianesthesia occurring with no, or only a transient, hemiplegia. Another disturbance of motility, however, then occurs,—hemiataxia,—probably a result of the anesthesia, especially the disturbance of the sense of position.

Sensory symptoms of irritation in the paralyzed or anesthetic members are not often complained of. *Pain* may occur from articular alterations resulting from inactivity, and only rarely as a result of trophic disorders. They occur especially in the shoulder joint. They may be due partly to muscular laceration caused by the pressure which the arm hanging limply like a dead mass to the trunk exerts upon the shoulder-muscles, or the pain may be a direct result of the cerebral disease, as has been shown on page 444.

Vasomotor disturbances are rare. At the onset the temperature of the paralyzed limb is often increased, while in later stages the skin on the paralyzed extremities may feel cool and is often of a cyanotic hue.

Certain articular alterations which develop, though only rarely, some weeks after the attack, and are combined with swelling, reddening, and pain in the joint, should be regarded as *trophic* disorders. Articular hemorrhages rarely occur (Obici).

Acute decubitus should also be included as a trophic disturbance, a decubitus developing very rapidly in the gluteal region of the paralyzed side some days or a week after the onset of the hemiplegia. According to Charcot's description, a flushing occurs first, followed by a spotted dark violet discoloration; next vesicles form, which become confluent; ulceration follows, and there develops an extended eschar. Some authors (Brown-Séquard, Hunnius) are inclined to bring the pneumonia, which occasionally occurs in apoplectics, into relationship with the cerebral disease, and to view it as a vasomotor-trophic disorder. Gastric hemorrhages are also regarded in this light by some (Charcot, Ebstein).

In describing the symptoms, we assumed that the hemorrhage occurred at the most common seat of the attack. It is natural that the picture must be modified considerably if the hemorrhage is in another location.

If the *centrum semiovale* is attacked, the hemiplegia in extensive hemorrhages is a direct or indirect focal symptom; smaller ones may not induce any resultant symptoms.

If the hemorrhage is located in the *central ganglia* of the *left* hemisphere or in the left internal capsule, a circumscribed focus may even produce a transient aphasia; the more extensive the hemorrhage is, and the nearer that it approaches the speech centres, the more marked and stabile is the aphasia.

The manifold phenomena produced by the varying localization of the cerebral hemorrhage need not be considered further here. Cortical hemorrhages, which are, on the whole, rare, may, when the motor region is affected, cause unilateral spasms, both tonic and clonic.

Only ventricular and pontile hemorrhages in addition to the above deserve special consideration.

Primary ventricular hemorrhage occurs very rarely. Of more frequent occurrence is the breaking through of an extravasation from a neighboring part of the brain (generally first into the lateral ventricles) and gradually filling all the ventricles. This produces severe symptoms: the disorder of consciousness deepens; if consciousness had been free or had again cleared up, a new apoplectic stroke follows; the paralysis extends to all four extremities; convulsions come on (unilateral on the, at the beginning, normal side, or generalized), more often a rigidity of the muscles of the hemiplegic side or in all four extremities. Generally retardation of the pulse, decrease of temperature, and marked respiratory disorders come on. Before death, which occurs in almost all cases, and, indeed, within twenty-four hours, the pulse becomes small and frequent; the pupils, contracted at the beginning, dilate and become fixed; and cyanosis and asphyxia come on.

In pontile hemorrhages consciousness may remain intact. The paralysis affects, as a rule, both sides of the body, and is combined with articulatory and deglutitory disorders. The pupils are generally contracted, a symptom that is already of importance during the coma; they may also, however, be dilated and fixed. Unilateral and generalized convulsions, and not rarely trismus, come on. The temperature tends to rise quickly and markedly to 104° F. or over; respiration is disturbed (Cheyne-Stokes breathing, simple retardation or acceleration and irregularity).

Pontile hemorrhages may also cause less severe symptoms (compare the chapter on acute bulbar paralysis). In a case observed by Elsholz, the paralysis confined itself to a number of cranial nerves. If in hemorrhage of the medulla oblongata death does not result immediately, severe respiratory and circulatory disorders develop in combination with the symptoms of bulbar paralysis.

Differential Diagnosis.—It is of the utmost importance in cases of apoplexy to make a correct diagnosis. Many and to some extent great difficulties must be overcome. It may be confused with conditions of simple loss of consciousness, with epileptic sopor, with hysteric conditions, with syncope, with uremic coma, and with other toxic forms of disturbance of consciousness.

Simple syncope is generally easily excluded; for in this disease consciousness is generally not completely abolished; the described signs of coma are absent; the attack, also, does not, as a rule, last long; the chief point for the diagnosis is, however, that the cardiac action is impaired; the pulse is small and often intermits. In a fainting spell the individual looks pale and stuporous, but not comatose.

An *epileptic attack* itself has no similarity to an apoplectic one; the soporous condition following or replacing it may, however, when the anamnesis is lacking, cause difficulty. If convulsions have preceded it, biting of the tongue, or a scar on this organ from a previous attack, indicates the epileptic nature of the seizure. If the coma forms an equivalent for the epileptic stroke, the facial pallor is noticeable. Unilateral symptoms are almost always missing in attacks of true epilepsy. Finally, epilepsy occurs, as a rule, in youthful persons.

A *paralytic* attack may completely resemble an apoplectic one: the differentiation can be made only from the anamnesis or from its further course. The same is true of apoplectic attacks in multiple sclerosis. One should always think of these symptomatic seizures when a young person with a sound cardiac and vascular apparatus is seized with a transient attack. *Hysteric* conditions of apparent or real loss of consciousness should hardly give rise to any confusion. The pupillary reflex remains intact, also almost always the corneal reflex; the same is true of the deep reflexes. The pulse-rate may be accelerated; it is, however, never markedly retarded. The facial expression generally indicates that the patient is under the control of an hallucination or some abnormal emotion. The temperature is neither increased nor decreased to any extent. Finally, it is at times possible by ovarian pressure or by psychic suggestion to relieve or to modify the attack.

Uremic coma cannot be diagnosed from a urinary examination alone, as albuminuria also accompanies an apoplectic stroke. A microscopic examination of the urine is often more satisfactory. The presence of edema or of albuminuric retinitis, and the knowledge that other symptoms of uremia have preceded the coma (convulsions, amaurosis, vomiting, asthmatic conditions, etc.), are points of much importance in the differentiation. The temperature is almost always subnormal. Uremic coma almost never develops suddenly. It often commences with vomiting and convulsions. Since, in atrophy of the kidney, we have the conditions which give rise to hemorrhage as well as to uremia, one must be chary about making a diagnosis during the coma. *Diabetic coma* is generally easily recognized from the result of the urine examination, the general constitutional condition, and the aceton odor to the breath. Attacks of *saturnine encephalopathy* are generally combined with delirium and convulsions; the anamnesis and the other signs of chronic lead intoxication rarely give a chance for an error in diagnosis.

Severe *alcoholic intoxicating conditions* can be recognized from the spirituous odor to the breath, the nature of the vomit, the tendency to delirium, and the motor restlessness which generally exists. It should, however, be remembered that cerebral hemorrhage not rarely follows an alcoholic debauch. In acute *morphine intoxication*, the pupils are contracted *ad maximum*.

Acute hemorrhagic encephalitis may commence with some disorder of consciousness, but marked premonitory signs usually precede it (headache, psychic defects, etc.), the temperature is generally raised, and the coma is not, as a rule, so deep as in apoplectic attacks, the reflexes and tendon phenomena being intact.

The question whether *cerebral hemorrhage* or *cerebral softening* is present is one of much practical importance. It cannot always be definitely answered. Cerebral hemorrhage is rare before the fourth decade, while embolus occurs often in youth, and thrombosis almost entirely in the aged. Heart disease renders embolic softening very probable; atheromatous disease of the heart should make us immediately think of thrombosis, though an embolus may also arise from an atheromatous heart. Finally, disease of the cerebral vessels occurring at the same time may produce cerebral hemorrhage. If a simple hypertrophy of the left ventricle is present, and especially if accompanied by nephritis, it is, as a rule, cerebral hemorrhage. If an acute articular rheumatism has preceded it, even if the heart does not show signs of disease, embolus should be thought of, as the endocarditis may have been cured. If the face is reddened and swollen, the pulse full, strong, slow, and tense, we are justified in diagnosing cerebral hemorrhage. We should, however, remember that the face may also be pale and the pulse soft and feeble in cerebral hemorrhage. The coma is, in general, deeper and of longer duration in cerebral hemorrhage than in encephalomalacia.

If a strongly marked atheromatous degeneration of the peripheral is found, cerebral softening is most probably the condition present. Syphilitic endarteritis also leads to thrombosis more often than to vascular rupture. Prodromes—paresthesia, temporary conditions of weakness in the later paralyzed side, etc.,—preceding the insult by weeks, rather indicate softening from thrombosis; while in embolus all prodromes (on the part of the brain) are generally absent; they are also often lacking in cerebral hemorrhage. Psychic weakness, especially, often precedes the onset of the thrombosis by a long time, and, on the other hand, is also a frequent accessory or resultant phenomena of it.

Repetition of the apoplectic insult indicates, in the first place, encephalomalacia. Infarcts in other organs (lungs, kidney, etc.) are particularly indicative of embolus.

A marked rise or depression of the temperature in the attack indicates hemorrhage. In ulcerative endocarditis, embolus may, however, be accompanied by fever and chills.

If the paralytic symptoms are present, if the insult has run its course or is missing entirely, the preceding details are still, to some extent, of value. The following should, however, be particularly inquired into. If unconsciousness has not occurred, and the other phenomena indicate a circumscribed lesion, it is extremely possible that it was a softening process. This view is not to be altered if a transient loss of consciousness or a vertiginous attack has preceded. Improvement beginning soon after the attack and then rapidly progressing renders the hemorrhagic nature of the process probable.

Prognosis.—(1) *In the Attack.*—An apoplectic attack may produce immediate death. The danger to life is based on the extent of the hemorrhage, its seat, and its breaking through into the ventricles.

The duration of the coma indicates to a great extent the size of the blood-clot. If it lasts over twenty-four hours, life is endangered, even though an awakening from it has been, in exceptional cases, noticed after two days A marked and constant sinking of the temperature is also suspicious, likewise an excessive rise (to 104° F. or more). The gradual onset of a continually deepening somnolence (ingravescent apoplexy) is also a bad omen. This is even to a greater extent true of acute decubitus. Conjugate deviation points to a larger focus, though it is in itself not indicative of danger to life. Cheyne-Stokes breathing is a bad sign. Nephritis makes the prognosis worse.

If symptoms indicating a rupture into the ventricles come on, the prognosis as to life is poor. Hemorrhages into the pons generally, and into the medulla oblongata almost always, take a fatal course; the appearance of bilateral paralytic symptoms is also always a bad sign.

Death may occur here, as in primary ventricular hemorrhages, in a few minutes. Another danger for apoplectics is the onset of *pneumonia.*

(2) *In the Paralytic Stage.*—If hemiplegia or another symptom-complex has developed as a result of the hemorrhage, it should be determined whether a retrogression will occur, whether recovery or improvement can be expected. Here also the degree and duration of the apoplectic attack are of value. The more incompletely this is developed, the better are in general the chances. A small hemorrhage may, however, be sufficient to completely divide the motor conducting tract when its seat is directly within it. It should therefore be ascertained whether the hemiplegia and the corresponding phenomena are *direct* or *indirect* focal symptoms. If the hemiplegia continues with unaltered intensity during the first month, or if only a trace of movement returns to the affected lower limb, it is almost certain that the hemiplegia is a direct focal symptom, and that only slight improvement can be expected. If a certain degree of motility returns within the first few days, and improvement gradually continues, it is a very good sign. Increased deep reflexes do not, without something further, make the prognosis worse. If, however, the first signs of contracture come on, the chances for complete recovery are no longer present.

Aphasia, when combined with hemiplegia, and if the diagnosis of cerebral hemorrhage is certain, has on the whole a relatively good prognosis. The speech is generally regained, even if a long time is necessary for a complete restoration of the disorder. A partial aphasia may also remain permanently.

If hemianopsia is present and does not improve immediately in the first few days, it will remain a permanent symptom.

If the paralytic symptoms disappear very soon—within some days—after a stroke, paralytic dementia must also be thought of. A careful examination should, however, determine whether it is present or not. When the mental disturbances are not yet marked, the pupillary reflex being intact, and a characteristic speech disturbance is lacking, it may be difficult and even impossible to establish a certain diagnosis. Dementia following the attack and lasting a long time is an unfavorable sign.

If the hemiplegia is a direct focal symptom, the patient may learn to walk again, but it is at least three to four months before he regains this ability. If only a result of compression, it may retrogress within a few weeks, though generally some months are necessary for the restoration of the power of locomotion.

The life of a person who has recovered from a cerebral hemorrhage is also generally endangered for the future, in that the predisposing factors to a repetition of the seizure are present. On the other hand, I know—excluding children affected with cerebral paralysis—individuals who have lived twenty to thirty years with their hemiplegia, without a new attack having appeared. The observation of Ballet and Dutil, that old blood-clots form a breeding-place for bacteria, does not appear to be of much practical interest.

Treatment.—*Prophylaxis.*—This includes measures to prevent vascular diseases and to relieve or render harmless such disorders. We are, however, confined within narrow limits. In the first place, all alcohol and other poisons already mentioned should be avoided. Remedies to restrain the formation of miliary aneurisms are unknown to us. As obesity seems to favor the onset of cerebral hemorrhage, appropriate dietetic procedures are in place. Iodine preparations should be used to combat arteriosclerosis. Rumpf recommends a diet poor in calcium salts and the administration of lactic acid. If the conditions which give rise to hemorrhage are once present, everything should be avoided which produces a sudden increase in blood-pressure and causes a rush of blood to the head. (Compare with the preceding chapter.)

In the apoplectic attack, false measures may lead to disastrous results. Great caution and mature deliberation are here necessary.

If it cannot be definitely determined whether hemorrhage or softening is present, the measures used should be limited to the following: The patient should lie quietly, with the head slightly raised, and with unencumbered neck, in a room removed from all noises and intense light. If he has fallen to the floor, remove the clothing from his neck, and then carry the patient, whose head should be well supported, to bed, avoiding all concussion. The relatives or friends should be informed that the patient will probably awaken after some time, and that he will perhaps not be able to move the limbs on one side of the body. All information liable to cause worry should be withheld; only a member of the family or a nurse should attend to the patient, and guard him as much as possible from all excitement and from all bodily exertion. The person affected by the stroke should repress all attempts at active motion; he should not endeavor to move the paralyzed limbs. If deglutition is impaired, caution is necessary in the administration of nourishment, so that the patient does not cough. Defecation should be promoted by the use of clysters, and rendered easy by the administration of laxatives.

If the face is red and swollen and the pulse full and strong in the attack, and if the other symptoms are also indicative of cerebral hemorrhage, *venesection*, especially when the coma lasts a long time, should be made. This should be done at the usual place in the upper arm, or in a vein of the back of the foot. It is contraindicated by general weakness, and especially in cardiac weakness, when the pulse is small and intermittent, or when other signs indicate an encephalomalacious process.

Attempts should not be made to bring the patient out of the coma.

Traumatic cerebral hemorrhages have several times been operated upon, even when the subcortical region was the seat of the hemorrhage. The operation was reported to have been successful in the published cases.

If cerebral hemorrhage is present, an ice-bag should be placed upon the side of the head corresponding to the lesion. As this procedure, so far as we know, is harmless, it may be employed in encephalomalacia also.

In cardiac weakness, small quantities of wine or cognac are in place; also other excitants, as ether and camphor injections, may be used.

Alcohol is not permissible in the first stage of cerebral hemorrhage. It cannot, however, be entirely stopped in persons addicted to its constant use, and where the danger of delirium is a threatening one. Coffee and tea should also be forbidden. Milk, cocoa, lemonade, and non-carbonated waters are the proper drinks. In the first few days a fluid diet is preferable; after the third day easily digestible solid food may be given in small quantities. The physician must watch that no digestive disorder, and with it vomiting, occurs.

When there is no particular occasion for their use, drugs need not be administered in the first stage. The bromides should be given for any restlessness that occurs; an opiate or sulfonal and trional for severe headache or continued sleeplessness. If syphilitic endarteritis is suspected, potassium iodide and mercury are in place. The former is often prescribed also, in the hope of promoting absorption.

Decubitus must be prevented by cleanliness, proper position, etc., of the patient. A water cushion may even be necessary.

As to the *treatment of the hemiplegia*, the paralyzed extremities should be softly massaged, and passive movements should be carried out in the individual joints, especially in those of the arm, in order to combat any contractures and disorders due to inactivity; ten to twenty minutes are required for this procedure. If the patient is excited or exhausted by them, they should be shortened. After two to three weeks an electric treatment is also advisable. Direct galvanization of the brain is preferably avoided.

It is permissible to excite contractions in the muscles of the paralyzed extremities with a faradic current. As soon as a tendency to contracture is noticeable, the electricity should be confined, if possible, to the antagonists of the contracted muscles. The electric current should be used for five minutes daily for from four to six weeks; the treatment should be then discontinued, to be resumed later on and carried out for months. I have been able in a number of cases to satisfy myself of the effect of electric treatment. One hemiplegic was able soon after a sitting to write relatively well and rapidly, while before it he wrote slowly, laboriously, and indistinctly. Another, who had been paralyzed for years, became able to extend his foot. If the hemianesthesia appears prominently, it is advisable to stimulate the anesthetic skin with a faradic brush. Vulpian observed good results therefrom, as have I also.

Treatment of the paralyzed part of the body with the galvanic current is also justifiable.

At first passive, later active, gymnastic exercises are of benefit in this stage.

When should the patient get up? As soon as the first signs of movements are again apparent, the patient has the desire to attempt to walk. The physician may be easily led into allowing him to do so, in order to convince him of the progress made. Against one such attempt nothing can be said. The patient should, however, never be permitted to remain permanently out of bed. There is no doubt that these attempts at movement favor the onset of contracture. Even after the leg has almost recovered its motility, the patient should pass most of the day in bed. On arising, it should particularly be seen to that the hanging arm is well supported in an appropriate sling.

If cyanosis or a sensation of coldness in the paralyzed extremities is present, they should be well wrapped up, without exercising any pressure upon them. Massage and passive movements are the best methods with which to treat the articular alterations. Embrocations can also be used.

Subcutaneous strychnine injections do no good in hemiplegia.

Erben calls attention to the fact that it is advisable in certain cases (when the knee cannot be actively flexed) to have the patient step out with his paralyzed limb and then draw the sound one forward only far enough to bring this foot beside the paralyzed one.

It is not impossible that muscular transplantation, as described on page 158, will acquire some importance in the treatment of hemiplegia.

If the apoplectic attack has left behind an aphasia, appropriate instruction may improve this condition. (Page 467.) Water-cures do not accomplish much. All hot or cold baths should be avoided. Only baths of from 78° to 80° F. are advisable. These should also be used cautiously, at first only in the form of washings, half-baths or foot-baths, and not too early, never before the fifth or sixth week. The laxative mineral waters can be recommended, perhaps drink-cures at the resorts themselves (Kissingen, Marienbad, Homburg, etc.).

The whole manner of living should be altered with the object of avoiding all rush of blood to the head. (See the chapter on cerebral hyperemia.)

CEREBRAL SOFTENING (ENCEPHALOMALACIA).

The cause of cerebral softening is local anemia of the cerebral matter induced by closure of an artery. The cerebral region deprived of nourishment degenerates, which is shown by a softening, in the original sense of the word.

The occlusion of the artery is due to an *embolus* or a *thrombus*. Emboli generally originate in the heart (valvular disease, especially mitral stenosis, or cardiac weakness with the formation of a thrombus), more rarely from the aorta (atheroma, aneurism), and still more rarely from the pulmonary veins (ulcerative bronchitis, formation of cavities and pulmonary gangrene).

Only rarely are particles of tumors of the heart or lungs carried to the brain. Other cellular material may compose the embolic material, though we can ignore here these rare occurrences, including also the so-called echinococcus embolus.

An embolus may also be a disintegrated part of a thrombus, which exists in one of the larger cerebral arteries.

Thrombosis of the cerebral vessels almost always develops as a result of *senile atheroma* or *specific endarteritis*. A similar disease of the cerebral arteries may also result from chronic intoxication (alcohol, lead, etc.); or hereditary influences may predispose to it. According to our experience, those diseases of the nervous system which are accompanied by permanent disturbance in function of the cardiac and vascular nervous system, as cardiac neurasthenia and certain forms of the traumatic neuroses, may incite the development of atheroma. Continued emotional excitement may act in the same way.

We assume that some diseases increase the ability of the blood to coagulate, and may induce a thrombus with intact vessel wall; we include here the *acute infectious fevers*, *phthisis*, and the *puerperium*. It has been presumed that the presence of micro-organisms and poisons in the blood increases its power to coagulate. Others believe that the infectious disease first produces an endocarditis, and that from this an embolus occurs, which, after the healing of the endocarditis, or if it remains latent, is regarded as a thrombus. Marked *cardiac weakness* favors the occurrence of thrombosis or of embolus, as it tends to form cardiac thrombi. Retardation of the blood-current helps the formation of thrombi, but can hardly by itself produce them. If, however, from one of the preceding causes the coagulability is increased, a thrombus will form wherever the blood circulates slowly or in a current vortex (v. Recklinghausen), as in passing from a narrow lumen into a wider one.

Chlorosis and *leukemia* likewise favor the onset of thrombosis.

In CO poisoning, softening occurs, the genesis of which is not very clear. The softening involved the lenticular nucleus of both sides particularly often (Poelchen, Kolisko). Multiple thrombi were also found in the brain after extensive burns of the skin (Klebs).

Traumatic encephalomalacia, softening in the vicinity of blood-clots and tumors, and the rarely observed thrombosis in tubercular meningitis, will simply be mentioned here.

If the predisposing causes for embolism or thrombosis are present, emotion, especially terror, physical over-exertion, and parturition (particularly for embolus) act as exciting causes. Hemiplegia has several times come on during *narcosis* (Schwartz, Chipault) without it being able to be determined whether hemorrhage or softening was the cause.

Emboli generally lodge at the bifurcation of a large artery, most frequently in the middle cerebral artery and its branches, and favor the left side. The internal carotid and deep cerebral arteries are not rarely, and the vertebral, especially the left, is at times, the seat of emboli.

Thrombi may develop anywhere; but mostly involve the larger arteries at the base of the brain, generally the middle cerebral, internal carotid, basilar, and deep cerebral, or branches of these.

The embolus, which forms a firm, colorless clot adherent to the vessel wall, and is at times calcified, may cause a secondary clot in the neighboring parts of the vessel; these secondary thrombi are reddish in color and spongy. The thrombus tends in this manner to grow, and by its growth to clog the vessels branching peripherally from the occluded vessel. Emboli may disintegrate before necrobiosis occurs, and the particles be carried away by the blood-current. This is particularly the case with emboli containing specific material or micro-organisms. Thrombi may also become absorbed.

In specific endarteritis, which attacks particularly the chief branches of the circle of Willis, the vessel lumen may be obliterated for some distance.

Closure of the lumen of the blood-vessel produces softening of the corresponding cerebral area only when a collateral circulation cannot be produced. This is always the result when the affected arteries are terminal arteries, as in the branches supplying the white matter of the brain, or when the artery becomes obliterated to such an extent that the places of entrance of the lateral branches promoting collateral circulation are also clogged up. It happens, for example, that thrombosis of the internal carotid, which generally extends to the place where the anterior and middle cerebral arteries are given off, may produce severe and permanent results, while in ligation or in embolism the collateral circulation is generally established through the circle of Willis. In the cortex collateral nourishment is not so easily impaired as in clogging of the arteries of the brain-stem. In occlusion of the middle cerebral artery, for instance, softening may not involve the cortical region, or confines itself to a small area, while the part of the inner brain supplied by it is softened over a large extent.

If the closure of the artery of the fossa of Sylvius occurs before the giving off of the basal arteries, which pass to the interior of the brain, the entire lenticular nucleus, the anterior part of the optic thalamus, the subthalamic region, the central convolutions (for the most part), the insula, the operculum, and the third left frontal convolution undergo softening, according to Monakow. Closure of the posterior cerebral artery deprives the greater part of the occipital lobe, particularly the calcarine fissure and the cuneus, and also the posterior thalamus of its blood, though the extent of the softening depends upon the seat of the thrombus and the development of the collateral circulation.

Foci of softening are found in all parts of the brain; they seem to prefer the cortex, but this is because it has the largest surface. They also develop often in the deep layers of the centrum semiovale. The head of the caudate nucleus and the anterior part of the putamen also form favorite seats for it. They occur most rarely in the cerebellum.

The softening process itself does not occur immediately after the vessel has closed; thirty-six to forty-eight hours go by, exceptionally three to four days, before the disintegration and decreased consistency occurs, though swelling (serous saturation of the affected region) is said to occur immediately after the embolus.

If the occlusion is not complete, it may not extend farther, or necrobiotic (and indurative?) processes may come on, which do not pass over into softening. Such a genesis, has, for example, been ascribed to certain forms of lobar sclerosis.

We speak of *red*, *yellow*, and *white softening*. The color is directly dependent upon the amount of blood present. Red softening is observed particularly in cortical foci, because the gray matter is richer in blood. Marked fatty degeneration of the diseased tissue and, particularly, conversion of the coloring-matter of the blood into pigment produce yellow softening. Red softening always represents an early stage, yellow softening only coming on in a few weeks. A hemorrhagic infarct, in Cohnheim's sense, develops rarely, though small blood extravasations are not infrequently observed in the periphery of the focus of softening. In the white matter the area of softening is generally of a white or bluish-white color. The tissue is of a broth-like consistency, or even watery (lime-water-like). It contains myeline drops, detritus, and particularly numerous granular cells, which are the best means of distinguishing true softening from post-mortem maceration. After resorption of the fluid material cysts may form, though the softened focus may remain in its original condition for years.

The size of encephalomalacious foci varies from that of a pin-head or even smaller to that of the fist, may even take up the largest part of a hemisphere, as, for instance, in thrombosis of the carotid. I found the whole hemisphere softened in one case. Cicatrization is the general termination of cortical softening. In the neighborhood of small arteries a circumscribed perivascular sclerosis is often observed instead of softening. In atheroma, instead of a large focus, or in addition to it, we often find a large number of small foci, which are, in part, only recognizable microscopically.

Emboli occur most often in young and middle-aged persons, while thrombi, excluding the syphilitic forms, affect aged individuals almost exclusively.

Symptomatology.—Encephalomalacia, like cerebral hemorrhage, is characterized by transient and permanent, local and general symptoms.

Larger foci of softening are, especially when of embolic origin, inaugurated by an *apoplectic attack*. Emboli of small branches and thrombosis of even large arterial trunks may occur without any disorder of consciousness. The coma is generally not so deep as, and mostly also of shorter duration than, that accompanying cerebral hemorrhage. The temperature decrease is frequently lacking, while, as a rule, about eight to ten hours after the onset of the disease, a rise of temperature occurs. It does not mount very high, but may last a number of days. The fever at times only commences after some days. Higher temperature and chills only occur with septic emboli.

Embolus of a larger artery almost always produces *loss of consciousness*. It is due to the sudden circulatory disorder, which is not confined to the affected area, but extends more or less over the whole brain; to this is added a sort of shock (Trousseau, Wernicke), a mechanical injury to the brain in that the bloodless vessels collaborate and produce a local displacement of the surrounding tissue, etc.; we also, with Monakow, regard it as probable that mechanical stimulation excites the vasomotor centres by reflex action and produces thereby a general cerebral anemia. In place of coma a generalized or unilateral epileptic seizure, or even a sort of status epilepticus, may come on. Vomiting is also not an uncommon phenomenon. More rarely the embolus induces only a mild attack, in which a condition of somnolence with delirium manifests itself, symptoms which are thought to be due to reactive occurrences in the vicinity. Thrombosis of main arteries also may usher in an apoplectic attack, but this is more often missing here than in hemorrhage, or the unconsciousness is very slight. It also often happens in thrombosis that the paralytic symptoms precede the clouding of the consciousness, and that this latter is replaced by a condition of mental confusion, or a slight delirium lasting some days.

If the attack is lacking, as is the rule in occlusion of small arteries, and common in thrombosis of larger ones, the patient complains of headache and vertigo. *Prodromes* are more important here than in cerebral hemorrhage. Embolus, it is true, develops quickly without premonitory cerebral symptoms. Certain troubles, however, precede thrombosis for days, weeks, and even years, referable to a cerebral affection. They are the general results of atheroma or of specific endarteritis, and consist of headache, vertigo, often an impairment of the memory and of intelligence, conditions of mental confusion and excitement, not rarely combined with delusions, and especially a lack of ability to correlate matters with time and place; not rarely also repeated mild attacks of stupefaction due to temporary circulatory disturbances or to small foci of softening.

Emboli may precede the embolization in other organs (kidneys, spleen, etc.); of particular interest is the connection of cerebral emboli with the central retinal artery.

The typical form of paralysis produced by encephalomalacia is *hemiplegia*, which comes on in the same manner as in cerebral hemorrhage. A *step-like development* of the paralysis occurs, however, at times in senile atheroma, and more often in specific arteritis. A hemiparesis precedes the hemiplegia, or one limb, and after some hours or days the other, becomes paralyzed, or unilateral paresthesia precedes the paralysis and hemianesthesia.

The hemiplegia is often accompanied by *aphasia* when it affects the right side, and this is generally a direct focal symptom.

As the foci of softening confine themselves not rarely to circumscribed cortical regions, *monoplegia* with *aphasia*, or *aphasia* alone, or hemianopsia may form the residual symptom of this cerebral disease. As, however, the softening may produce *indirect* symptoms, and the clogging of the vessel lumen affects very often the middle cerebral artery, it can easily be seen that hemiplegia, alone or with the above symptoms, is almost always present in encephalomalacia. If it is an indirect symptom, if the focus of softening does not disturb the motor tract or the motor centres directly, it disappears in a short time. Hemiplegia may be accompanied by *hemianesthesia*, or this symptom may occur alone.

We speak of *temporary hemiplegia* when softening does not occur,—due either to the fact that disintegration of the clot occurs, or that it is carried away, or that a collateral circulation forms, or (as in lues) the nutritive disorder was only due to a marked contraction of the vessel lumen, which for a time amounted to a clogging of it. This hemiplegia disappears in some hours or days.

At times the symptoms do not correspond to the seat of the softening; they ridicule our laws of localization. It should, however, be remembered that numerous smaller foci occur scattered through the brain, in addition to the large one, many being only detectable by a microscopic examination.

Symptoms in Obstruction of the Internal Carotid.—If a collateral circulation is established, the hemiplegia rapidly retrogresses or is absent entirely (as in parturition).

If the vessels are too narrow to supply sufficient blood, or are lacking, or if the thrombus extends into the anterior cerebral artery and fossa Sylvii, it produces a very extensive softening, causing hemiplegia with deep, permanent coma, and generally soon ends in death. From obstruction of the ophthalmic artery (and centralis retinæ), a more or less severe visual disturbance may take place. In embolus of the carotid the phenomena vary, according to the seat of the clot.

Obstruction of the middle cerebral artery (trunk) produces complete hemiplegia with temporary or permanent hemianesthesia, and (in obstruction of the left middle cerebral artery) total or partial aphasia. If the obliteration confines itself to one of the branches, the phenomena modify themselves according as the softening involves the third frontal convolution, the anterior or posterior central, the parietal, or the first temporal convolutions. If it is an embolus, and the clot extends to the cortical branches of the central convolutions, unilateral convulsions often precede the onset of the paralysis. According to the extent of the region cut off, monoplegia (faciolingualis, faciobrachialis, etc.), a hemiplegia, a motor aphasia (with or without paralysis), etc., develops. Obliteration of the first branch of the left side causes motor aphasia with right faciolingual monoplegia; the arm may be paretic also. Closure of the second branch produces hemiplegia or faciobrachial monoplegia. Obstruction of the third and fourth, which supply the lower parietal lobes, results in alexia when the lesion is on the left side, and generally, also, in hemianopsia, and perhaps word-deafness. The last is the chief symptom when the fifth branch is clogged up, etc.

Embolism or thrombosis of the *anterior cerebral artery* (the first occurs rarely) may produce crural monoplegia and psychic disorders (?).

Obstruction of a branch in the white substance generally evokes hemiplegia (or hemianesthesia); if the softening does not extend beyond the caudate or lenticular nucleus or the optic thalamus, hemiplegia may not occur.

Closure of the *deep cerebral artery* results in *hemianopsia*, and. as a rule, in hemianesthesia, if a collateral supply is not established.

Monakow gives the following picture of thrombosis of this artery. If the thrombosis comes on, as it usually does, in steps, general phenomena, as vertigo, conditions of mental distraction, transient hemianopic disorders, and scotoma scintillans, with headache and temporary amblyopia, come on first; then follows a true apoplectic attack with a resulting hemiplegia, perhaps also with convulsive movements. After the disappearance of the general symptoms, the hemiplegia also passes away, as a rule, and there remains the *hemianopsia*, and, in left-sided lesions, sensory aphasia or paraphasia and amnestic aphasia. Hemianesthesia may also be present.

Consult the chapter on acute bulbar paralysis for the symptoms occurring in thrombosis of the basilar and vertebral arteries.

In some cases of arteriosclerosis a number of small foci of softening develop without any marked apoplectic seizure coming on. The paralytic symptoms, to a certain extent, slowly increase in intensity, so that their development and progress correspond more to the type of a chronic disease. Disorders of intelligence, as a rule, often spasms of laughing and crying, and, not rarely, bulbar symptoms, occur (see chapter on pseudobulbar paralysis). At any time, however, true attacks with typical symptoms may manifest themselves. In this *multiple encephalomalacia* a form of amnesia occurs, which may simulate an amnestic aphasia, without there being present the corresponding disease of the speech centre. These conditions are closely allied to senile dementia and may pass into it.

Differential Diagnosis.—The criteria already given should serve to differentiate softening from cerebral hemorrhage. We would call attention, in addition, to the fact that the face is generally not sunken or reddened in the apoplectic attack of encephalomalacia.

If the symptoms refer to a cortical lesion (aphasia, monoplegia), it is probably a case of softening.

Encephalomalacia may be difficult to distinguish from cerebral tumor if the first is preceded for a long time by prodromes; or if the tumor, after remaining latent for a certain time, suddenly produces paralysis. In cerebral softening, however, the typical signs of increased cerebral pressure are generally lacking,—*i.e*, choked disk, retardation of the pulse, and continued, progressive stupor. When, in the first, a slow pulse is present from atheroma of the coronary arteries, other symptoms generally give a clue to the disease. It should, however, be remembered that the presence of atheroma does not exclude cerebral tumor. Vomiting occurs rarely in thrombosis, or only at the onset; the exception to this rule being obstruction of the basilar artery, which again is revealed by other important signs. The convulsions occasionally occurring in embolism and thrombosis, even when unilateral, have not the typical course of cortical epilepsy.

For the differential diagnosis from cerebral abscess, compare this chapter with that upon the latter disease.

When encephalomalacia only presents general cerebral symptoms (headache, vertigo, stupor, insomnia, etc.), and no focal phenomena, it may be confused with the neuroses, especially with hysteria and neurasthenia. The constancy of the symptoms, their independence from self-observation, the signs of vascular disease, and the objectively noticed stupor, indicate encephalomalacia. An extensive softening also tends in time to injure the general health and even to produce marasmus.

Finally, it should be noticed that in some cases of hemiplegia no alterations in the brain are found at the post-mortem. This is certain in *tuberculosis*, *alcoholism*, *uremia*, *diabetes*, *lead poisoning*, *arthritis*, and *pneumonia*. I have observed it in *carcinomatous cachexia*, and have expressed the opinion that it is a *toxic focal disease*. There are, so far as I know, no absolute clinical signs enabling us to distinguish hemiplegia of this nature from the form due to organic alterations.

The prognosis as to life is good. Thrombosis of the basilar and carotid are the only forms in which there is a direct danger to life.

Long duration of the unconsciousness is not such an ominous sign in encephalomalacia: the patient may come to even after five or six days. If the coma is deep, however, it is a bad sign. The general condition and that of the heart should also be looked into. If the area of softening is not very extensive, life may last many years.

The prognosis of the paralysis is, in general, unfavorable. If it does not recede in the first two to three weeks recovery cannot be expected, as collateral circulation is restored within a few days, and the so-called indirect focal symptoms tend to disappear in from two to three weeks. On the whole, these paralyses are not as important in softening as in hemorrhage. Softening due to specific endarteritis is also not capable of retrogression. The prognosis is, in addition, dependent upon the location of the softening and the extent of the lesion.

Treatment.—Venesection is not advisable, though still recommended by some physicians. In the attack, the position of the patient, rest, and similar precautions require as much attention as in cerebral hemorrhage. Defecation should be attended to, though laxatives should not be used. If the heart's action is weak, excitants should be prescribed.

If lues is the original cause, potassium iodide and mercury must be given at once. Even if we cannot thus affect the softening, the vascular disease may nevertheless be stopped.

Concerning the treatment of the paralytic symptoms and the general dietetic measures, consult the preceding chapter.

CHRONIC PROGRESSIVE CEREBRAL SOFTENING.—A number of observations indicate that softening of the cerebral substance may take a slowly progressive course. Small and large foci of softening have been found in the brain without an obliteration of the artery supplying these regions. The symptoms were generally those of a gradually developing hemiplegia. General symptoms were entirely absent or confined to head-pains. Local muscular contractions preceded the paralysis in several cases. Pain and anesthesia have also been observed. The hemiplegia generally forms within some months, or it keeps on progressing for a longer period ; then follows a cessation, and death comes on from some intercurrent affection. Most of the persons were aged. In one case of this kind, which I observed, I found the cerebral arteries free, but the cervical carotid was thrombotic or closed through an obliterating arteritis.

ENCEPHALITIS (ACUTE NON-PURULENT FORM).

The term encephalitis is applied to various forms of cerebral inflammation. The cerebral form of multiple sclerosis is, for instance, called a disseminated encephalitis. The different forms of meningitis (traumatic, tubercular, epidemic, and pyemic) produce alterations of the cerebral matter—especially in the neighborhood of the affected meninges—which belong in the category of encephalitis.

In *hydrophobia* and *chronic chorea* pathologic conditions of this kind have also been found. The still-debated "encephalitis of the new-born" (Virchow) cannot be discussed here. Disseminated myelo-encephalitis is treated elsewhere (page 206). Purulent encephalitis will be given a separate chapter. These forms of encephalitis will not be discussed here, partly because they are spoken of at other places, partly because the encephalitis is not the prominent clinical part of the disease-process. The two most important and independent forms of encephalitis in question here are (1) *acute hemorrhagic encephalitis*, (2) *acute hemorrhagic superior polio-encephalitis*.

1. Acute Hemorrhagic Encephalitis.—This disease has been particularly studied by Strümpell, Leichtenstern, and Fürbringer. The cause, so far as we know, is always, or in most cases, *infection*. Cases of this kind have especially been noticed at the time of *influenza* epidemics, and were thought to have some connection with this disease. The disease may probably also develop after measles (Henoch), scarlet fever, pneumonia (Carré), and erysipelas. Encephalitis after whooping-cough (Neurath and others) and mumps (Putnam) has also been lately described, and though *post-diphtheritic hemiplegia* has once (Mendel) been asserted to be due to hemorrhage, and many times (Henoch and others) to embolus, it cannot be doubted that this paralysis may be due to hemorrhagic encephalitis. The anatomic investigations of Krauss and the clinical observations of myself and Thiele point to this. A report by Coester seems to indicate a relationship between encephalitis and erythema nodosum. In other cases, in which a connection between a definite infectious disease has not been proven, a relationship with *cerebrospinal meningitis*, to a certain extent an encephalitic abortive form of this disease, has been assumed. The disease has also been observed in connection with *ulcerative endocarditis*.

It is not improbable that in some of these cases we are dealing with an independent infectious disease. In the central nervous system of influenza patients who died with cerebral symptoms, Pfuhl found influenza bacilli, and Nauwerck was able to detect them in the foci of influenza encephalitis. The secondary infection and the potency of the bacterial poison may also play a part.

The etiologic importance of *traumata* in non-purulent encephalitis has not been decided. Some observations, however, show that encephalitis may be the result of cranial injuries, even of ordinary contusion of the head. It is questionable whether the traumatic form is identical with the infectious type. The possibility of the trauma producing a lesion which serves as a breeding-place for the bacteria should be remembered.

Symptomatology.—The disease develops acutely, is usually even foudroyant, and affects *youthful*, previously healthy persons. Children and young girls are most often affected. It has been observed several times in *anemic* girls.

Without prodromes, or after one to two days of headache, vertigo, depression, or irritability, the patient becomes *stuporous*, *unconscious*, and the unconsciousness rapidly increases to *sopor*. A *chill* sometimes precedes the onset of the disorder of consciousness. Though it resembles an apoplectic condition, the coma is rarely so deep, and the pupillary reaction to light and the tendon phenomena are almost always present, generally also the skin reflexes. The temperature does not fall, but is immediately increased, or rises later on, and paralytic symptoms are lacking or only appear in a few cases at the beginning of the disease. Stiffness of the neck may come on during the attack. General convulsions occur rarely. Restlessness, mental confusion, and delirium may accompany the stupor. Respiration is generally accelerated; Cheyne-Stokes breathing has been observed several times. The pulse is, as a rule, abnormally frequent; it may, however, especially in the first stage, be slowed. Enlargement of the spleen was observed in only a few cases.

The coma may deepen and death come on in from twenty-four to seventy-two hours—preceded by a gradually increasing rise in temperature, especially marked near the time of death—without the patient awakening from his coma, as I have seen it in one case. It may run a more protracted (even subacute) course, and I have seen cases ending in death only after twenty days. *Remissions* generally occur then, the fever is also apt to be remittent; the sensorium especially may clear up temporarily, and paralysis may come on.

It may be a *monoplegia, hemiplegia,* or *aphasia,* according to the seat of the disease; the latter has been observed by me in a number of cases. Cortical epilepsy and hemianopsia, as in an observation of Fürbringer, rarely belong to the focal symptoms. In some cases I found *optic neuritis.* More rarely other cerebral nerves, as the abducens, were affected.

Not so often, but still in a large number of cases, the pons, medulla oblongata, and most rarely the cerebellum, were affected. Inflammatory processes were noticed in these parts of the brain by Leyden, Etter, and Eisenlohr, and described as *acute bulbar myelitis, inferior acute polio-encephalitis,* acute encephalitis, or bulbar poliomyelitis. I have seen several cases of this kind followed by recovery.

In another case of mine, *cerebellar symptoms, hemiataxia, nystagmus,* and *optic neuritis* occurred. It ended in recovery.

The pathologico-anatomic basis is an *acute inflammatory process* in the brain of an especially *hemorrhagic* character. This is generally confined to a circumscribed area, not rarely to symmetrical regions of the cerebrum. It has been found in the centrum semiovale, cerebral cortex, and especially often in the central ganglia. As a rule, therefore, the cerebrum is affected. The same process may occur, however, in the brain-stem, particularly in the gray matter lining the aquæductus Sylvii, in the pons, and in the oblongata. It has been found most rarely in the cerebellum. The affected tissue, upon macroscopic examination, appears strongly hyperemic, covered with spots resembling flea-bites, generally swollen and moister than normally. Only in a few cases were the gross alterations not pronounced enough to be visible to the naked eye.

Microscopically the small arteries and capillaries were found dilated, turgescent with blood, which had also flowed into the vascular sheaths or, after laceration of these, into the tissues; an infiltration of white blood-corpuscles, and, in old cases, granular cells and proliferated glia cells were also found. The nervous elements show evidence of irritation and degeneration. When we consider that a complete restitution is possible, even in cases where the symptoms indicate, for instance, an extensive break in conduction in the pons, it can be assumed that in these cases which tend to recover the anatomic alterations are also slight and do not pass beyond the first stage.

Focal diseases, which anatomically do not differ from encephalomalacia, but which cannot be referred to closure of an artery, also occur.

It is almost certain that infantile cerebral paralysis is often the result of an acute encephalitis, which was localized in the motor cerebral regions (v. Strümpell). The occurrence of acute encephalitis in early childhood has been particularly studied by von Ganghofner, Fischel, and Raymond. There is no doubt that its forms vary according to the character and extension of the process. In some cases, as in one of Fischel, it was a diffuse process extending over both hemispheres. Raymond believes that this diffuse encephalitis may pass into a diffuse cerebral sclerosis (see page 210). Finally, disseminated myelo-encephalitis is not rarely noticed in childhood (see page 206).

A *sinus thrombosis* may be combined with the encephalitis (observations of Siemerling and myself, Bücklers, Nauwerck, etc.). I found in several cases a marked ventricular exudate, meningeal hyperemia, etc. A combination of encephalitis with acute anterior poliomyelitis has also been observed (Lamy, Redlich, Beyer).

As to the other organs, swelling of the spleen, nephritis, parenchymatous degeneration of the myocardium, and the like, have been noticed.

Diagnosis.—A diagnosis of acute hemorrhagic encephalitis must be made with caution. It is difficult and often impossible to distinguish it from acute serous meningitis, from sinus thrombosis, and from meningitic-like symptom-complexes (pseudomeningitis) (compare page 477) following the infectious diseases. (The appropriate sections should be consulted for the differential diagnosis.) Encephalitis differs particularly from these conditions in that in it the focal symptoms develop, as a rule, early, and become more and more the main symptoms.

The Prognosis of hemorrhagic encephalitis is grave. In cases with a rapid development, deep unconsciousness, and high fever, the course is probably generally a lethal one. On the other hand, the cases observed by myself and those described by Fürbringer, Fraenkel, Freyhan, Thiele, and others, show that recovery is not uncommon. I have seen recovery in a case of encephalitis which was confirmed by the anatomic examination made later. Convalescence may last for weeks, months, and even years. Recovery with a defect remaining also occurs; in fact, it is the rule, if we include here Strümpell's form of infantile cerebral paralysis. The disease appears to intermit sometimes (own observation, Dinckler).

Treatment.—There is not much to be said. It is certainly necessary to give the patient complete rest and careful attention. He should be placed in a room free from noise and bright light, and every emotional excitement should be avoided. Cold compresses, or an ice-bag upon the head, and venesection or leeches, are the therapeutic measures to be recommended at the beginning. The latter are contraindicated in anemic persons. We have no satisfactory experience concerning the influence of drugs, though salicylates, quinine, and antipyrin should be in place. In one case, in which recovery took place, in addition to bloodletting, *calomel in large doses*—to the development of a stomatitis—was used. Hot foot-baths may be ordered in lingering cases. The after-treatment is identical with that described for cerebral hemorrhage.

2. ACUTE HEMORRHAGIC POLIO-ENCEPHALITIS SUPERIOR.—Wernicke first described this clinical picture, which was further amplified by Thomsen, Boedeker, and others; Gayet, however, described a case of this disease before Wernicke had named it.

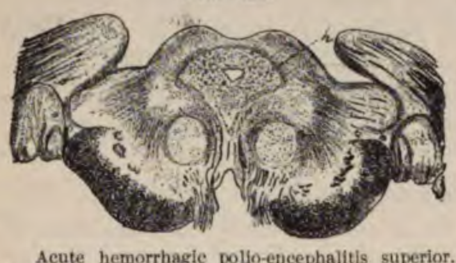
In cases of this type the acute hemorrhagic encephalitis confines itself to the ependymal gray upon the *floor of the third ventricle* and of the *aqueduct of Sylvius*. This type is treated of separately because its etiology and symptomatology differ somewhat from that of the others. Chronic alcoholism is the most important causative factor. Most of those affected were whiskey or brandy drinkers. It occurred once after poisoning from sulphuric acid.

Other poisons (meat-, fish-, bologna-toxines, etc.) may, perhaps, also cause it. Recent observations (Gayet, Uhthoff-Oppenheim, etc.) teach that this form of encephalitis may also *follow infectious diseases*, particularly *influenza*, and differs from the previously described encephalitis in difference of localization only. We will first present the clinical picture of the form described by Wernicke, Thomsen, and others.

The disease has an acute onset and takes, as a rule, an acute course, ending generally in death in from eight to fourteen days. A few cases have terminated in recovery. After signs of chronic alcoholism have been present for some time, or headache, vertigo, and vomiting for some days, or even without any prodromes of this nature, a disorder of consciousness in the shape of *delirium* or a simple *somnolence* with restlessness comes on. Rigidity of the neck occurred in a few cases, also sleepiness and apathy. At the same time *paralysis* of the *ocular muscles*—generally associated paralysis—develops, which may increase to an almost complete *ophthalmoplegia*, though a few muscles, as the levator palpebræ superioris and the sphincter iridis, are often spared. Optic neuritis may also occur. Nystagmus has also been frequently observed. A disorder of gait, reminding one of *cerebellar ataxia*, may also be present. Weakness, tremor, and ataxia in the extremities may also manifest themselves. Articulation has often been impaired. In a few cases facial paresis, in others hemiparesis, has been observed.

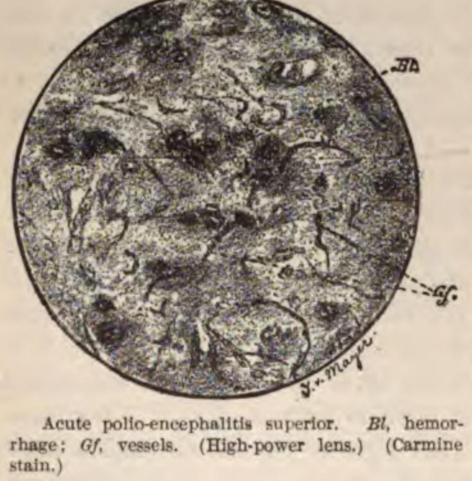
The deep reflexes are normal or exaggerated; they are rarely abolished. The temperature is almost always normal or subnormal. Fever is uncommon. The pulse is generally accelerated and becomes particularly small and frequent *sub finem vitæ*. Tachypnea and other respiratory disorders also develop. The disease takes a peracute or acute course and generally ends in death in from eight to fourteen days, though a protracted course and termination in recovery may occur.

FIG. 225.



Acute hemorrhagic polio-encephalitis superior. *h*, location of the encephalitic process. Spotted appearance due to the numerous foci. (Carmine stain.)

FIG. 226.



Acute polio-encephalitis superior. *Bl*, hemorrhage; *Gf*, vessels. (High-power lens.) (Carmine stain.)

In the cases examined so far, a hemorrhagic inflammatory process of the type described above was found, confined to the gray matter of the third ventricle, of the aqueduct of Sylvius, and at times extending to the ependymal gray of the fourth ventricle. Instead of a hemorrhagic inflammation, a degeneration of the corresponding nerve nuclei was found in one case.

In addition, the affection often extends to the white matter, and is analogous in many respects to poliomyelitis, with which it is often combined. On the other hand, it is certain that polio-encephalitis not rarely develops during a polyneuritis; just as a neuritis of certain cranial nerves may combine with the former.

In other cases, belonging here, the disease followed *influenza* or some other *infectious disease*, or no cause could be determined. In these cases it does not always have a stormy development, but extends over weeks or months. Stupor and every sign of a constitutional disease may be absent. In most of these cases, the process—so far as can be determined by the clinical signs and the few post-mortem examinations (Kaiser)—extends to the nerve nuclei upon the floor of the fourth ventricle. That is, a *polio-encephalitis superior* and *inferior*, and even a *polio-encephalomyelitis*, was present. Uhthoff and I saw a case in which dysphagia and disturbance of speech developed with paralysis of the ocular muscles, and which terminated in recovery. Similar observations have been made by Etter, Goldflam, and others.

The *focal symptoms* are the chief signs of the disease. They indicate a disease-process extending rapidly from the nuclei in the midbrain to those of the pons and oblongata. The phenomena of *ophthalmoplegia* and *bulbar paralysis* are variously grouped and combined in this disease. It generally takes a descending course, though the opposite also occurs. The *constitutional symptoms* correspond to those described at the beginning of this chapter; but they may be very slight or entirely absent. It is, however, the rule for the affection to be combined with increased temperature. The course is acute or subacute, at times progressing in steps. As, however, the anatomic process is not confined to the gray matter, but also extends at times diffusely over the pons and oblongata, the clinical phenomena may often represent a diffuse disease of this region, and all the clinical pictures which we will describe elsewhere as being produced by focal diseases of the pons and medulla oblongata may occur. The various forms of hemiplegia alternans, particularly, are produced by this disease.

Polio-encephalomyelitis causes a paralysis extending more or less symmetrically over a part of the motor nerves of the brain and cord. The affection of the cranial nerves produces the clinical picture of ophthalmoplegia and glossopharyngolabial paralysis; the spinal disease that of a diffuse or circumscribed, generally atrophic, spinal paralysis. Now this, now that component of the symptom-complex predominates. An electrical examination generally gave only quantitative decrease of excitability, or a partial reaction of degeneration. When the white matter is also involved, the clinical symptoms indicate it (sensory disorders, etc.).

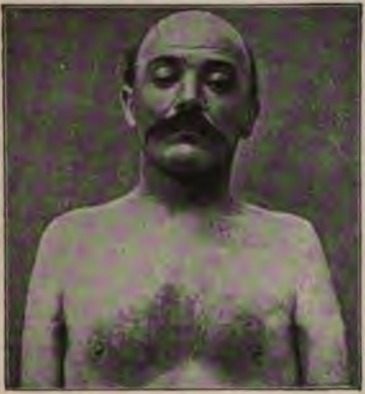
In most of the cases described (Rosenthal, Seeligmüller, Guinon, Parmentier, Kalischer, and others), the course was a *subacute* or *chronic* one.

Recovery has occurred in several subacute cases. The more chronic cases will be discussed at other places. See chapter on Ophthalmoplegia.

A diagnosis should only be made after a careful and complete examination, as allied clinical pictures occur without being traceable to material alterations. They are probably toxico-infectious conditions without an anatomic basis. Several times, instead of the expected tissue disease, a local invasion of micro-organisms was found (Eisenlohr, Seitz).

The two chief forms of encephalitis described above—Wernicke's and Strümpell-Leichtenstern's — are not easily separated from each other, as transitional and mixed forms occur, and both may also be present together.

FIG. 227.



Facial expression in polio-encephalitis superior and inferior. Facies Hutchinsonii. (After Guinon and Parmentier.)

CEREBRAL PALSY OF CHILDHOOD (HEMIPLEGIA AND DIPLEGIA SPASTICA INFANTILIS; INFANTILE CEREBRAL PARALYSIS).

This form of cerebral paralysis, of congenital origin or acquired in childhood, is not particularly characterized in its pathologic anatomy, but it is its clinical signs and its course which make it an independent disease. It rarely occurs in fetal life, more often *intra partum;* the phenomena appear mostly, however, in the *early years* of life, be it from the fact that it is only acquired in extra-uterine life, or that the symptoms of the congenital condition only appear some time after birth. One can therefore, with Sachs, distinguish prenatal, intrapartum, and acquired paralyses, though this differentiation is not always possible.

Our knowledge of the **etiology** is still imperfect. *Heredity* does not seem to play an important part, though I have seen the disease in mother and daughter; on the other hand, there are hereditary and family diseases of the nervous system which seem to be closely allied to this affection.

Traumata (injuries to the gravid uterus) seem to be an important cause of this disease in fetal life; psychic excitement is also blamed.

Syphilis of the mother, or hereditary syphilis, seems to have been a cause in some cases (Erlenmeyer, Osler). Of great importance are the injuries which act intra partum. *Premature birth, difficult delivery* with a narrow pelvis, difficult delivery of the after-coming head, twins, asphyxia of the new-born,—these are the conditions which often give rise to the disease. Forceps delivery is also blamed, although it does not appear to be as much the use of the instrument as the factors which render its use necessary. These traumata produce *meningeal hemorrhages*, which affect the meninges over the motor zone and evoke lesions of the cortex (Sarah McNutt). Hemorrhage is especially due to the veins being torn away before entering the sinus (Virchow).

Pushing of the parietal bones against each other is said by Kundrat in ordinary cases of labor to be able to cause a compression of the longitudinal sinus, by which the entrance of blood into the sinus is made difficult, and sometimes the veins entering it are torn away.

The *extra-uterine* etiologic factors are, particularly, infectious diseases. In a relatively large number of cases the affection develops during or after the acute infectious diseases. *Measles* and *scarlet fever*, with nephritis or endocarditis, most frequently precede it; but whooping-cough, variola, pneumonia, etc., may also cause it. It is said to have followed vaccination several times. Probably the bacteria act through the vascular system upon the brain; it cannot be easily determined to what extent *embolic processes* are in play, though emboli (with endocarditis) have been several times found in the region supplied by the middle cerebral artery.

Many observations render it probable that not a few cases of infantile cerebral palsy are caused by an infectious encephalitic process,—an acute non-purulent encephalitis, affecting particularly the motor region of the brain (cortex and medullary part, more rarely the central ganglia). This acute encephalitis of the motor region is, according to Strümpell, who, as did Benedict and Vizioli before him, brought it into analogy with acute anterior poliomyelitis, the typical basis of a typical form of cerebral palsy of childhood. Raymond claims to have found in a case of this kind a pathogenic bacillus in the diseased cerebral region. Gowers considers that an arterial or venous or sinus thrombosis is often the starting-point of the disease; others, again, believe in a hemorrhagic origin. *Cranial traumata* can also promote its onset in extra-uterine life. Fright has likewise been given as a cause.

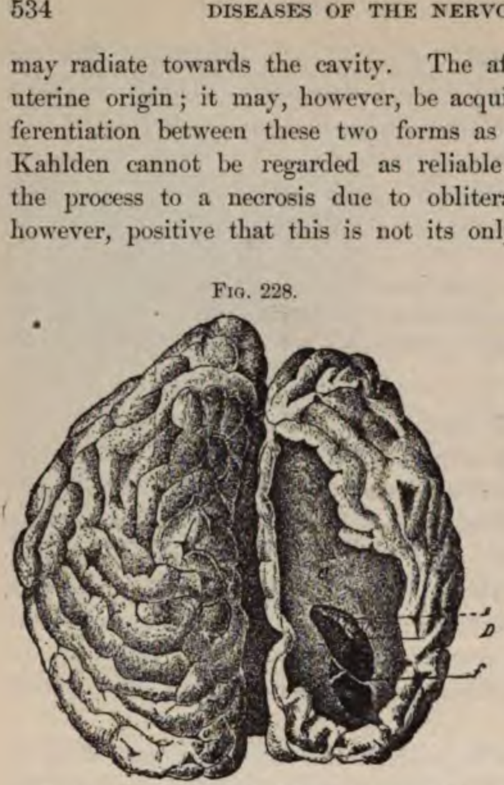
Pathologic Anatomy.—There is no single anatomic basis. In some cases a focal disease of the cerebrum was found,—a *focal softening*, one or more *cysts*, mostly of hemorrhagic origin, a cicatricial atrophy and *induration*, a *defective formation* of the cortex. (Fig. 228).

The latter condition, called porencephaly (Kundrat, Heschl), occurs on one or both sides, and most often in the central region; it appears as an irregular, funnel-shaped retraction of the cerebral surface, generally communicating with the ventricle. The convolutions bordering upon it may radiate towards the cavity. The affection is generally of intra-uterine origin; it may, however, be acquired post partum. The differentiation between these two forms as made by Kundrat and von Kahlden cannot be regarded as reliable (Beyer). Kundrat ascribed the process to a necrosis due to obliteration of blood-vessels; it is, however, positive that this is not its only mode of origin.

Several times have developmental anomalies of a peculiar kind been noticed in the cerebral cortex: a delicate folding of the cortical gray substance similar to the convolutions of the vermis cerebelli (*microgyria*). (Fig. 229.) Figs. 230 and 231 show the microscopic alterations in this developmental inhibition.

Similar anomalies are often found on the cerebral meninges, particularly thickening and cystic formations.

FIG. 228.



Porencephalic defect of the left hemisphere with atrophy. The cavity passes at *D* into the lateral ventricle, where the choroid plexus, *c*, is visible. (After Ferraro, reproduced from Starr.)

The affection is rarely confined to a circumscribed focus but extends diffusely over the whole or the greater part of a hemisphere, which is *in toto* smaller on account of the general atrophy (Fig. 228). This causes a chronic inflammatory process, a sclerosis. In some cases no focal disease, but only a general induration, has been found, the so-called *lobar sclerosis* (Bourneville, Richardière, Jendrassik and Marie).

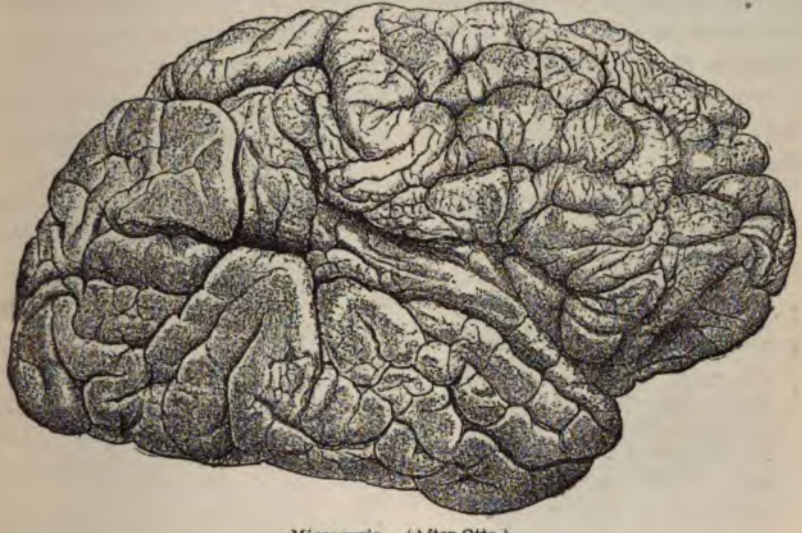
In hypertrophic sclerosis, excrescences are formed in the brain-substance. *Hydrocephalus* is often found in conjunction with the above alterations. A simple *cortical agenesis*, characterized particularly by microscopic alterations,—defective development of the nervous elements,—is occasionally noticed (Sachs).

The *motor zone* is generally affected, but rarely alone. The process is a diffuse one. The focus at times is found at other places; for instance, in the central ganglia. In a case of Bischoff's the thalamus was involved, in another the cerebral peduncle.

Porencephaly has also been found in other places besides the motor region; for instance, by Moeli and Richardière in the occipital lobe and by Monakow in the cerebellum. Naturally, different clinical symptoms would be found in such cases. Congenital blindness, deafness, cerebellar incoördination, etc., are probably due to such alterations.

Our knowledge of the *initial lesion* is very incomplete. We can regard it as definitely settled that hemorrhage in the brain-substance and meninges (McNutt), embolism, thrombosis with secondary encephalomalacia, encephalitis, and meningo-encephalitis may be the cause. If these disorders develop in childhood and affect the motor centres (or conducting paths) the symptoms of spastic infantile hemiplegia will be produced.

FIG. 229.



Microgyria. (After Otto.)

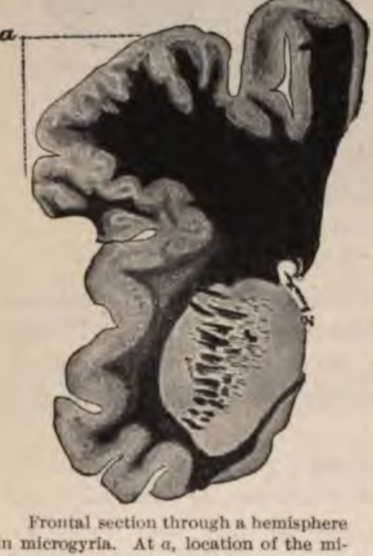
At a later period it is by no means always possible to recognize the nature of the original disorder, as certain conditions, as induration and atrophy of the hemispheres or a greater part of them, seem to be the result of all of the above-mentioned alterations. All harmful conditions affecting the infantile brain act unfavorably upon the motor conducting paths and tend to inhibit their development. Premature birth is also supposed to do likewise.

Symptoms.—The initial stage can be studied only in postnatal cases. In these cases the following is often the course: the child, from a few months to three years old, becomes sick with *fever*, *vomiting*, *stupor*, *delirium*, and general or, from the first, unilateral *convulsions*, and following these, or after repeated attacks of these spasms, the paralysis, a *hemiplegia*, comes on.

This initial stage lasts from one to several days, or it may extend over weeks. It is rarely completely absent. The paralysis occurs then suddenly (apoplectic), or after generalized or unilateral spasms. It is exceptional for the paralysis to be separated by a long interval from the convulsive stage. It may, however, be present at birth and only noticed later, or perhaps at once, and becomes more marked the more the voluntary movements on the sound side develop. In typical cases, *improvement* occurs in some weeks or months. A certain degree of movement returns, so that the individual learns to walk again, and is able to use the arm to some extent. Later on, however, certain characteristic disorders become noticeable, due to *muscular contractions* and *muscular spasms.*

The hemiplegia involves the arm, leg, face, and hypoglossus of one side. The least constant is the glossal involvement. The facial muscles are usually only partially paralyzed; when at rest, an asymmetry may be lacking, appearing, however, in laughter or crying. The motor symptoms of irritation in the face tend to be more marked than the paralysis. The arm is generally more paralyzed than the leg.

FIG. 230.



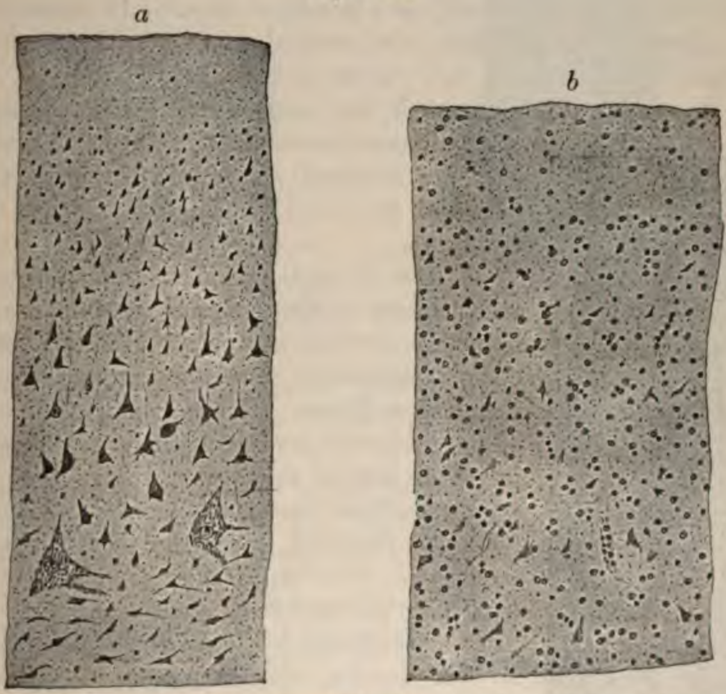
Frontal section through a hemisphere in microgyria. At *a*, location of the microgyria. Condition of the gray matter of the brain and of the subcortical fibres in this developmental anomaly.

At the onset of the recurring motility, or even earlier, the muscular rigidity or *contracture* becomes noticeable. This may be stabile,—that is, continually of equal intensity, producing a fixation of the limb in a definite position: the upper arm drawn towards the trunk, the lower arm flexed, the hand markedly flexed (Fig. 232), or over-extended, the fingers flexed at all joints and sunken into the hand; or the hand may be extended at the interphalangeal joints, and over-extended even to subluxation. The leg is generally slightly flexed at the knee-joint, and the foot is found in an equinus position.

Or we find a *spastic innervation of the muscles* accompanying voluntary movements: every effort at movement causing tonic contractions or spasms instead of simple movement. As a rule, a certain degree of muscular contraction is permanently present, but it is increased on attempts at active movement, and forms a considerable inhibition to them.

There are forms of infantile cerebral paralysis in which the rigidity forms the only symptom, while there is hardly any paralysis present. With the increased muscular tonus we find increased deep reflexes; increased knee-reflexes are almost always present; ankle clonus is, however, not so frequently found. The intensity of the muscular spasticity is by no means the same in all the muscles involved in the paresis; it may be more marked in the leg than in the arm; it may be prominent in the shoulder muscles, while the hand and fingers enjoy free passive movement, sometimes even to an abnormal degree, so that the fingers may be over-extended in the carpophalangeal and interphalangeal joints. (Fig. 233.)

FIG. 231.

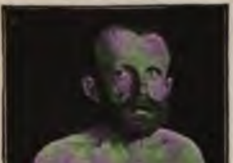


Ganglion cells of the cortex of the motor cerebral region: *a*, in the normal brain; *b*, in microgyria. (Nissl's stain.)

Voluntary or associated movements (*mitbewegungen*) are almost regular concomitant symptoms of hemiplegia of childhood. They are, however, rarely as pronounced as in Westphal's case, where the excursions of the involuntary movements were almost as great as those of the actively moved extremity.

Motor symptoms of irritation, *athetosis* or hemiathetosis, and hemichorea, are characteristic symptoms of spastic infantile paralysis, more so than those above mentioned, and have been already described. (Compare page 440.) They are present in most cases, at times slightly; on other occasions so marked that they dominate the clinical picture. Athetosis is more frequently observed than chorea, though the distinguishing criteria are not absolute. I have seen cases in which the phenomena in the upper limb resembled chorea, and those in the lower limb were more like athetosis. It may be present in congenital cases from birth, or it may develop as a primitive athetosis in extra-uterine life, without being preceded by a paralysis. As a rule, however, it associates itself earlier or later to the hemiplegia. It is permanent, although it may vary in intensity or accompany only voluntary movements. The hand and fingers are involved the most; the patient can only open the closed hand or shut the open hand slowly, etc.

FIG. 232.



Case of infantile spastic hemiplegia of the left side.

A form of tremor similar to an intentional tremor occurs rarely. In some cases the paralysis disappears entirely, and only a slight athetosis or a slight tendency to associated movements reveal the previous disease. Headache and sensitiveness of the cranium to percussion in certain areas was present in some of my cases.

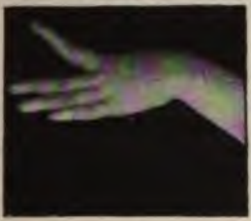
Hypesthesia has been occasionally noticed (Oulmont, Gaudard, Raymond). I found it in a recent case four weeks after the onset of the paralysis; also once in an adult who had cerebral palsy of childhood combined with hemiathetosis. In this case the special senses on the same side were impaired, and vasomotor disorders were present, so that a combination with hysteria could not be excluded. In several cases I found, with intact sensibility, Wernicke's tactile paralysis (see page 444). As the hemiplegia, however, was a congenital one, or one of early childhood, it was probable that the tactile memory pictures were not acquired by the paralyzed hand. Sometimes severe pain in the diseased side is complained of. Hemianopsia was found in only a few cases (Freud). An *aphasia* is often combined with the hemiplegia (especially hemiplegia dextra), which disappears again, sooner or later. It is generally a motor aphasia.

If the child could not yet speak, the development of speech is delayed by the hemiplegia.

Paralyses of the ocular muscles are an uncommon complication. Atrophy of the optic nerve may also be a symptom. (König.)

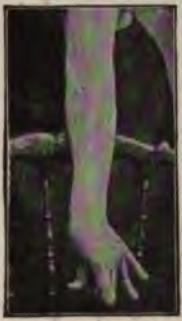
The paralyzed extremities are generally arrested in growth. A *decrease of muscular volume* (without qualitative alterations of electrical excitability) may also be present. A slight facial hemiatrophy has also been observed in a few cases. Muscular hypertrophy is at times combined with the athetosis.

FIG. 233.



Over-extension of the fingers at the interphalangeal joints from infantile spastic hemiplegia.

FIG. 234.



Hyperextension of the thumb from athetosis in infantile spastic hemiplegia.

Alterations of the bones of the cranium, depressions, fissures, decreased circumference, etc., are inconstant symptoms (Peterson and Fisher, Breschet, Meschede, and others).

In a girl with infantile spastic hemiplegia of the right side I found a defect of the left frontal bone, over which a cerebral murmur could be easily heard; at the same time there was a large telangiectasis on the nose. Kalischer has recently shown a preparation of a child with infantile spastic hemiplegia which showed a telangiectasis on the scalp and an analogous neoplasm on the cerebral surface, especially in the membranes over the motor zone. Atrophy of the scrotum, abnormal atrophy or hypertrophy of the mammæ, arrested growth of the little and ring fingers (Féré), and other developmental anomalies are occasionally found upon the paralyzed side.

Of the symptoms still undescribed, the most important are *imbecility* and *epilepsy*.

Epilepsy is found in one-half to two-thirds of all cases. It may develop immediately after the initial convulsions or after an interval of months, years, and even longer. There is generally a period of from one to two years between the onset of the hemiplegia and that of the epilepsy. The epilepsy rarely corresponds in all particulars to the genuine form. The convulsions generally confine themselves to the paralyzed side or are more strongly marked upon this side and commence here. They may be entirely similar to *cortical* epilepsy. If confined to one side, consciousness may not be lost. The initial scream, the biting of the tongue, the sedes inscii are generally absent. The attack is sometimes not as complete as it is in true epilepsy. Each attack comes on spontaneously or is incited by some emotion.

I treated a girl with cerebral palsy of childhood in whom an attack could be provoked by frightening her; for example, by dropping a chair to the floor behind her. Her father claimed the attacks could sometimes be cut short by a "counter-fright."

These attacks may be very frequent and intense. The status epilepticus also occurs.

In some cases of infantile cerebral palsy, the paralytic symptoms disappear and all that remains to indicate the disease is an epilepsy, twitchings in the formerly paralyzed side, a temporary paralysis following the attack, etc.

In a large percentage of cases, the mental development of the child becomes impaired, all transitions from slight feeble-mindedness to complete *idiocy* occurring. Marked irritability and maniacal conditions are also not uncommon. The imbecility occurs particularly in cases combined with epilepsy. Bourneville and König have shown the many relations between idiocy and cerebral palsy of childhood.

The intelligence need not, however, become impaired.

Freud particularly has made the attempt to group the different cases of cerebral palsy of childhood and to classify them into different types.

The congenital forms should, if possible, be differentiated from postnatal cases, but this cannot always be done. The differentiation can be carried out to a certain point. In congenital cases, the symptoms come on immediately after birth or shortly afterwards. They are for a time (about a year) progressive in character. At one time the paralysis, at another time the motor symptoms of irritation are most marked. The child learns to read late, the development of speech is also arrested. Very often all four limbs are involved.

Strümpell has attempted to differentiate a form which he regarded as analogous to infantile spinal paralysis. This was always a disease acquired in early childhood. A marked initial stage similar to the course of an acute infectious disease, with convulsions, hemiplegia, retrogressive course, improvement of motility after some months, and later appearance of motor symptoms of irritation, and very often epilepsy, characterize this form.

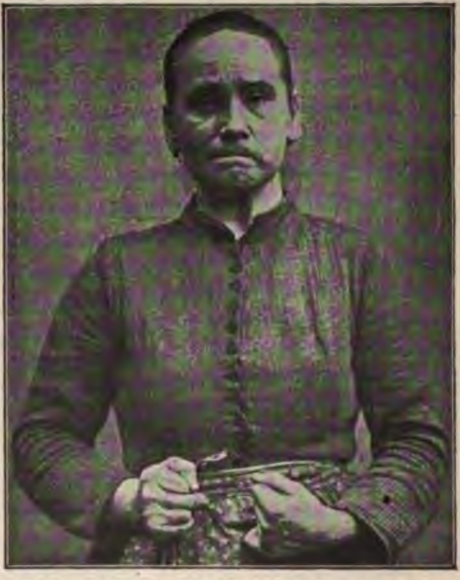
Freud has called attention to the fact that some cases develop gradually, and commence not with paralysis, but with chorea or athetosis. This form, called by him *choreatic paresis*, is also distinguishable by a late onset, not until the third to the sixth year; by the absence, as a rule, of aphasia, epilepsy, and dementia; by slight contractures; and by the prominence of the chorea and athetosis.

In some cases the spasticity and weakness extend to the leg of the sound side. It is not a rare occurrence for the athetosis to appear in the limbs of the normal side, though to a less degree.

These cases form the transition to an important group (studied particularly by Freud) in which the hemiplegia is bilateral, all four extremities being affected by the paralysis and spasms (*infantile spastic diplegia*).

Freud classifies these cases into four groups,—(1) general cerebral spasticity (Little's disease; his view is, however, too narrow,—see page 143); (2) paraplegic rigidity; (3) bilateral hemiplegia; (4) general chorea and bilateral athetosis. Even he, however, acknowledges that this differentiation cannot be closely carried out, transitional and mixed forms occurring more often. Groups 1 and 2 have been described on page 143.

FIG. 235.



Facial expression and pose in athetotic-spastic diplegia.

As a rule, the spasticity and weakness are most marked in the lower limbs, while the choreic-athetotic movements dominate in the upper extremity. A general athetosis or chorea may, however, also be present. Bilateral athetosis forms at times a progressive disease occurring in later life (Albutt, Oulmont, Andry). In many cases of infantile spastic diplegia, articulation is also impaired; and I have several times observed distinct signs of glossopharyngolabial paralysis with dysarthria, and even with mutism, in these complicated cases of bilateral infantile paralysis. A habitual subluxation of the lower jaw also occurs. The facial expression and whole appearance (*habitus*) of these cases has something characteristic about them (Fig. 235).

The intelligence is impaired more often than in simple hemiplegia, but not constantly, while epilepsy occurs more rarely. The speech disturbance may also be due to choreic involvement of the articulatory and respiratory musculature. In this form, the truncal and nuchal muscles are often involved, so that the child is unable to hold up its head.

Most of these cases of diplegia are of intrapartum or congenital origin, general spasticity (in Freud's sense) being mostly a birth-palsy (abnormal position, narrow pelvis, asphyxia neonatorum), and paraplegic spasticity often the result of premature birth. That both hemispheres are diseased is evident. In addition to the remarks made for Little's disease, we can add here that there are cases of bilateral athetosis in which no cerebral disturbance can be detected. A single case of this kind with evidence of a spinal lesion upon autopsy (Eisenlohr) cannot, however, be used to generalize from.

Diagnosis.—The differential diagnosis does not offer much difficulty. Confusion with *infantile spinal paralysis* is hardly possible: the spastic, non-degenerative character of the paralysis, the combination with chorea, athetosis, etc., the involvement of the facial—these are sufficient diagnostic points. Some observations (Lamy, Beyer) indicate that both affections may be combined; I have seen a clinical case of this character. It is more difficult to recognize and to classify the diplegic types. I have seen cases of this kind which on superficial examination resembled simple chorea; but its early onset (soon after birth), its resistance to treatment, and the spastic weakness of the lower limbs which developed later, enabled me to make a correct diagnosis. In the very rare cases of brachial monoplegia, be it that it existed alone from the beginning, or formed a residual symptom of a hemiplegia, there is some similarity to obstetric paralysis, but the latter, if it persists, is a flaccid degenerative paralysis.

It is certain that this disease may be obscured by symptoms of epilepsy, the paralysis and other symptoms having disappeared entirely, while the spasms continue. The character of the epilepsy, the anamnesis, the detection of a slight athetosis, or marked associative movements, are symptoms which will often lead to a recognition of the disease.

The anatomic basis cannot be definitely diagnosed in individual cases.[*] Porencephaly also, notwithstanding Brissaud's attempts, cannot be definitely diagnosed.

Prognosis.—Cerebral paralysis of childhood is an affection which shows little tendency towards recovery. There are a certain number of cases in which improvement progresses far enough for us to speak of an almost complete recovery: a certain awkwardness in one hand or a slight tendency to athetoid and associative movements may be the only residual symptom. Most cases, however, are incurable: a certain degree of motility is regained, but it remains only a partial one, because contracture and athetosis set in. The latter may render the extremity completely useless, the free movements of hands and fingers particularly being abolished.

Contracture, athetosis, and chorea, when once completely developed, rarely recede entirely. Improvement of the paralytic symptoms is to be expected in the first year; it may, however, continue for years. This description of the prognosis refers to the complete clinical picture of the cerebral paralysis, not to the acute disease causing it. It should not be forgotten that some of the processes enumerated (hemorrhage and acute encephalitis) are capable of complete recession.

If from two to three years have gone by without any epileptic attacks, it is somewhat improbable that they will appear. Epilepsy may, however, develop ten years or more afterwards. In mature years the spasms become rarer and are said to disappear entirely in the fifth decade. Death may, however, occur at any time in the epileptic status.

It can generally be determined in the first year whether the intelligence has been much weakened. Retarded development of speech and of mental capacity do not in themselves denote a permanent defect. Aphasia is a symptom with a good prognosis, as it almost always disappears.

The patient may reach an old age, and is sometimes, to a certain extent, capable of doing work. One of my patients, for instance, is a driver, using the affected left hand only as an aid to the right; another is a newsboy, etc.

The prognosis of Little's disease is relatively favorable. The symptoms may gradually decrease in intensity, though improvement does not advance to recovery.

Treatment.—There is rarely an opportunity to treat the disease at the beginning. The symptoms would generally demand an antiphlogistic treatment, ice-bag, local bloodletting, etc.

Our therapeutic endeavors must generally be directed against the paralysis, contracture, athetosis, and epilepsy. The results of treatment are not brilliant, but by the use of the *faradic or constant current, massage, and passive movements*, a certain degree of improvement can be secured in many cases. Bromides should be used for the epilepsy, though their effect is not reliable. To quiet the chorea and athetosis, Sachs has the restless hand carry an iron plate.

It appears to be possible that plastic operations of the tendons (page 158) could be used here also to counteract the paralysis and contracture (Eulenberg).

On account of the untrustworthiness of symptomatic treatment every attempt to influence the disease directly should be welcomed.

Surgery has entered this field of cerebral diseases. As meningitic thickening, meningeal and intracerebral cysts have been found in many cases, it was expected that excision of the thickened membranes and emptying of the cysts would do good. It was particularly hoped that cases of epilepsy would be benefited. The results are, however, hardly conducive to further experimenting, though some (Starr, Sachs, and Gerster) have recently again insisted upon such operations, and have given definite indications. In an especially severe case of this kind, many years ago, in which the epilepsy was so severe and the attacks so frequent that the condition was almost unbearable, I was induced to allow an operation.

A girl, twelve years old, had suffered since her fourth year with right hemiplegia and hemiathetosis, together with spasms which commenced on the right side. The skull was sensitive on percussion in the left temporo-parietal region. Sonnenberg trephined it in this region, and a cyst, the size of a plum, was found in the cerebral membranes. This was emptied, and the brain not touched. The wound healed nicely, the spasms also became less in number and decreased in intensity. The athetosis seemed to decrease somewhat. This improvement did not, however, last long, and after a year the girl died in status epilepticus. The autopsy revealed an old extensive focus of softening extending deep into the brain in the region of the central convolutions and the lower parietal lobe (probably of meningo-encephalitic origin), and also an atrophy of the entire left hemisphere. It need hardly be said that the evacuated cyst played but a minor part in this case.

Lannelogue's operation need not be described here.

There are a number of hereditary diseases of the nervous system which seem to resemble the different forms of diplegia very much, and yet, on the other hand, correspond to the type of certain spinal diseases. Most of the observations of this kind refer to a congenital or early-developed spasticity in several members of the same family (Schultze, Bernhardt, Kojevnikoff, Newmark, Erb, and others). Cerebral symptoms, particularly strabismus, speech defects, and nystagmus, are also found. Sachs has described a type which probably belongs here, which occurs almost exclusively in Jewish children; its chief symptoms are the following: idiocy, spastic paralysis of all four extremities (perhaps a flaccid one also), and blindness from optic atrophy with peculiar alterations of the macula lutea, which had been previously noticed by Tay and others. The children die early. A degeneration of the cortical cells, especially the pyramidal cells, was found, though Sachs does not regard the term *agenesis corticalis* as expressing entirely the anatomic basis; he proposes, therefore, from the clinical symptoms, the name "amaurotic family idiocy."

I saw a combination of spastic-ataxic paralysis with pes cavum in several members of the same family.

We know little of the pathology of these diseases. They are so closely allied to diplegia that they cannot be easily separated from them. On the other hand, they are very similar to the hereditary form of spastic spinal paralysis (see page 142).

The neuron theory warns us not to lay too much stress upon the separation of cerebral and spinal diseases of the motor areas from each other.

CEREBRAL ABSCESS.

A collection of pus in the brain is the result of a *purulent encephalitis* due to *micro-organisms*. It is either of *traumatic* origin or the infectious material comes from a near-by or distant collection of pus. Only rarely do bacteria directly circulate in the blood and produce a primary purulent encephalitis.

In a large percentage of cases the abscess is due to a trauma. A slight scalp wound is sufficient to give access to the germs, but generally complicated fractures have occurred, or a slow suppuration has preceded it, or foreign bodies have passed into the brain. *Simple cranial contusion is not able to produce cerebral abscess.*

The formation of the abscess immediately follows the injury or occurs some days later; it is then generally a superficial cortical abscess, which, on account of its frequent combination with meningitis, is of no particular clinical interest. Or between the injury and the formation of the abscess lies an interval of relative or complete good health; the abscess develops then, as a rule, in the inner part of the brain, in the gray matter, and generally in the part covered by the parietal or frontal bone, as this part of the head is the most frequently injured. It should be remembered that the *trauma may have been slight and long forgotten* when the symptoms of brain abscess appear. Cases, however, have been observed in which ten to twenty or even thirty years have elapsed between the injury and the onset of cerebral symptoms.

A suppuration on the cranium often furnishes the septic material which is carried to the brain. *Chronic purulent otitis media* is the chief cause, as one-half or at least one-third of all cases are due to this. The otitis is acquired mostly in childhood, especially after acute infectious diseases, and lasts years or decades before the brain becomes involved. Suppuration of the tympanic cavity and its accessory cavities, especially the cells of the mastoid antrum, caries of the petrous bone, as well as *cholesteatomata*, are the conditions which endanger the brain.

The tympanic membrane is almost always perforated, and *purulent otorrhea* is present, or has been for months or years. Acute exacerbations of chronic suppuration and arrest of the evacuation of the pus from granulation is particularly dangerous. Only in a small number of cases did the cerebral inflammation follow an acute otitis media (with or without perforation of the tympanic membrane). The aural disease may, however, be cured before the cerebral symptoms come on.

The parts of the brain adjacent to the bony walls of the auditory organ are those which become infected. These are the temporal lobes, from the upper part of the tympanic cavity (being separated from it by only a thin surface), or from the roof of the mastoid antrum, and the cerebellum from the mastoid process, the cells of which were almost always involved when pus was found in the cerebellum. The suppuration may occasionally extend from the labyrinth into the cerebellar fossa. Generally the bones are diseased through to the dura (Körner); at times a fistula led from the abscess through the thickened, coalescent, or purulent infiltrated meninges to the foci in the bone; as a rule, however, they are separated by a more or less thick layer of cerebral tissue. It generally does not, therefore, result from continued extension of the suppuration from the bones to the brain-matter, but the micro-organisms pass from the cortex more or less deeply into the gray matter, by way of the perivascular lymph spaces, or pass from thrombotic veins of the pia into the brain-tissue. The pus may also pass along the *acoustic* and *facial*. There is often, in addition to the intracerebral suppuration, an extradural one in the middle or posterior cranial fossa, or in both, and in addition generally a sinus thrombosis. This also forms by itself a frequent complication of abscess.

Only in a few cases does the cerebral abscess originate in carious processes on other bones (ethmoid, sphenoid), suppuration in the nasal cavities, orbits, infection after the extraction of polypi from the nose, etc. Recently the observations of cerebral abscess of nasal origin have increased considerably (Kuhnt, Dreyfuss). Purulent diseases of the bronchi and pulmonary tissue (*bronchiectasis*, *pulmonary gangrene*, *pulmonary abscess*), also empyema, are the chief causes of *metastatic* cerebral disease (Virchow, Biermer, Gull, and others). We find multiple abscesses in these conditions, as also in pyemia, *ulcerative endocarditis*, etc. In one hundred cases of pulmonary gangrene Nähter found purulent foci in the brain eight times. Böttcher discovered pulmonary pigment in cerebral abscess. Thrush is said to have produced metastatic cerebral abscess (Zenker, Ribbert), also *actinomycosis*. In a case of otitis, after influenza and pneumonia, pneumococci were found in the pus of the cerebral abscess. *Phlegmonous* processes may also infect the brain from distant regions of the body (purulent ostitis, arthritis, etc.). This is, however, rare.

There are some cases in which none of the causes mentioned can be detected. In some of the cases described as "idiopathic" some factor, especially a trauma, may have been overlooked. In a few cases *tubercle bacilli* were found in the granulation tissue covering the inner wall of the abscess, as also in the pus itself (A. Fraenkel). In other cases a relationship between it and an infectious disease, especially *epidemic cerebrospinal meningitis,* must be assumed, be it that this preceded the abscess formations, or that the abscess developed at the time of an epidemic (Strümpell). Other infectious diseases, as *erysipelas, influenza, measles,* etc., may cause purulent encephalitis through the transmission of an otitis; it is, however, not improbable that the bacilli of these diseases may *directly* reach the brain and produce a "primary" cerebral suppuration (Martius).

Cerebral abscesses of otitic origin always have their seat in the same side of the brain, in most cases in the *temporal lobes* or in the *cerebellar hemisphere.* In seventy-six cases statistically reviewed the abscess was found fifty-five times in the temporal lobes, thirteen times in the cerebellum, four times in the cerebrum and cerebellum, twice in the pons, and once in the cerebral peduncle.

They are found most often in the posterior inferior part (third temporal convolution, fusiform gyrus), not rarely extend to the occipital lobe or are found here alone. In the cerebellum it is generally the anterior external section which is affected. The *traumatic* forms lie in the neighborhood of the injured cranial region,—that is, in the frontal and parietal lobes. Those of *nasal* origin occur almost always in the frontal lobes. *Metastatic* forms favor the region supplied by the artery of the fossa of Sylvius, especially the left.

Traumatic and otitic abscesses are mostly *solitary.* In about fifteen to twenty per cent., there were found more than one focus (two near each other, or one in the occipital lobe and another in the cerebellum). Metastatic ones are generally *multiple.* Bergmann found in a case of pyemia after gangrene of the leg, more than one hundred in the brain.

The foci are of varying size, from a pea to that of the fist. The solitary are, on the average, the size of an apple to a walnut; the multiple generally smaller.

The pus has generally a green or greenish-yellow color, often a fetid odor, and sometimes contains degenerative products of the cerebral tissue.

Older abscesses are, as a rule, enclosed in a hard capsule, the formation of which often commences in the first weeks after the suppuration comes on, but takes one and a half to two months to become firm. This inclusion does not mark the cessation of the process, as further growth from disintegration of the cerebral substance is possible, also a breaking through the capsular wall. On the other hand, the encapsulated purulent focus may remain latent in the brain for years until spontaneously or from some external cause it becomes active again. The tissue around the abscess is generally softened and edematous. In the pus are found the streptococcus pyogenes, the staphylococcus pyogenes aureus, albus, and citreus; in one case it contained the pneumococcus (Sahli), and in several, tubercle bacilli.

The abscesses often *break through into the ventricle* (particularly those of the temporal lobes) or to the meninges, so that a diffuse suppurative meningitis develops.

Very rarely it breaks through externally into the nasal orifices, tympanic cavity, or even through the temporal bone. Extradural suppuration, sinus thrombosis, meningitis, and thrombophlebitis are the chief complications. In this way cerebral abscess may lead to pyemia, embolic pulmonary abscesses, etc. Cerebellar abscess is often combined with hydrocephalus.

It is improbable that cerebral abscess can be spontaneously reabsorbed. Inspissation and partial calcification do not even remove the danger.

Symptomatology.—The symptoms are dependent, (1) upon the suppurative process, (2) upon the cerebral disease, (3) upon the original disease. The etiology influences the symptomatology so much that the clinical pictures of traumatic, otitic, and metastatic abscesses differ in many points. It is, however, advisable to set up a picture that will embrace all and yet indicate differences dependent upon the etiology.

In many cases the abscess produces cerebral disease of acute onset with an acute or subacute course, in others, it takes a chronic course and can then be divided at times into four stages,—(1) the initial stage, (2) the latent, (3) the stage with pronounced symptoms, (4) the terminal stage. The first is rarely observed; in general, only the anamnesis gives us any knowledge of the second stage, and only the third and fourth, often combined into one stage, come under the physician's notice.

The following description refers mostly to the phenomena of this period. It is particularly true of otitic abscesses that the initial and latent stages are often lacking or not observed, as the symptoms are not very different from those of the aural disease. The disease, as a rule, therefore, commences with its manifest or terminal stage and takes an acute or subacute course,—that is, extends over a period of several weeks or sometimes over one to three months.

The symptomatology of *traumatic* purulent encephalitis is similar to that of purulent meningitis of the convexity. In a few days or from one to two weeks after the injury headache comes on, or the headache present since the injury increases in intensity. In addition, vomiting, fever, stupor, general convulsions, restlessness, mental confusion, occasionally delirium, also occur. If the motor cortex is affected, signs of cortical epilepsy and monoplegic paralyses may come on. The stupor increases to coma, and in this condition death follows in a few days or after two to three weeks, or a remission occurs which is naturally only of short duration.

There is a great similarity to purulent meningitis; only when the symptoms appear after one to two weeks, do not have such a stormy course, remit or even intermit, can we assume that a cerebral abscess is present. Slowing of the pulse and low temperature also indicate it. Early and marked cortical focal symptoms also render it probable. *Deep* cerebral abscesses—*i.e.*, the late traumatic and otitic ones, etc.—are of greater clinical interest. In the former, the abscess, as a rule, remains latent for some time, weeks, months, and even years,—*i.e.*, does not reveal itself by any or only by indefinite symptoms,—when suddenly or gradually the signs of cerebral disease appear.

Upon closer investigation and observation, the latency is often seen to be only partial; headache occurs, which is not looked upon as being of any importance, or an increased temperature, which is referred to the existing otitis or to some slight constitutional disorder. In other cases, spasms appeared which were regarded as epileptic or hysteric. At times, emaciation, loss of strength, or psychic anomalies, as apathy, melancholia, and mental distraction, induce the physician to fear cerebral abscess. These symptoms are, however, not always present, and intervals of good health may intervene between the cerebral attacks.

The most constant and the earliest of the active symptoms is *headache*. It may be slight or very painful. In many cases it is particularly intense at one place, corresponding to the seat of the abscess. At this place percussion will also produce a more or less severe pain. The headache is increased by everything which produces a rush of blood to the head or cephalic congestion. *Vomiting* is a common symptom, being rarely absent in cerebellar abscesses.

A symptom of great diagnostic value is the *fever*, which is usually slight and inconstant, rarely reaches a high degree or lasts very long, and in some cases is entirely absent. Subnormal temperature is not an uncommon phenomenon. The head-pain increases with the rise in temperature. *Chills* occasionally occur, but are not frequently repeated. *Slowing of the pulse* is a frequent and important symptom, sinking even to thirty beats a minute. Respiration may also be retarded and irregular.

Generalized epileptoid convulsions may occur at any time, but are not a constant symptom. Consciousness is rarely uninvolved. *Stupor* is generally present, which may increase at any time to *sopor;* in many cases restlessness, irritability, and mental confusion come on, or severe *delirium* occurs. Apathy and depression sometimes dominate the mental scene. The psychic picture may alter itself in a very short time. In not a few cases are seen *optic neuritis* and what Knies calls *obstructive neuritis,*—a choked disk, which is not so marked as that found in tumor, and is not constant. Emaciation, cachexia, more rarely icteric discoloration of the skin, may be among the symptoms present. The gastric functions are almost always disturbed, loss of appetite, obstipation, *fœtor ex ore,* etc., occurring. The symptoms described above are produced partly by the suppuration, and are partly due to general disease of the brain and to increased cerebral pressure. There are other symptoms which depend upon an involvement of a definite part of the brain, and which, therefore, have the significance of focal symptoms.

Focal symptoms are absent in some cases, particularly when the abscess occurs in a silent part of the brain,—*i.e.,* in a part of the brain, a lesion of which does not cause any local symptoms. These include abscesses occurring in the frontal lobes (particularly the right), in the right temporal lobe, and to a certain extent those of the cerebellum. Further, it should be remembered that circumscribed foci of pus may occur in every part of the brain, without producing focal symptoms. I believe myself not far wrong, however, in asserting that focal symptoms are present in most cases, but are not always recognized or valued correctly.

Abscesses of the left temporal lobe produce the characteristic phenomena of word-deafness. I, as well as others, have been able to make a local diagnosis through this in many cases, and to give the seat at which pus was sought for and found. In the first successfully performed operations in otitic cerebral abscesses (Schede), sensory aphasia was the symptom which indicated the place for the operation. It was formerly not always correctly recognized, but was regarded as a "psychic disturbance." It is rare to find a complete word-deafness; more often it is a *partial,* an *amnestic aphasia,* a *paraphasia,* or, as I have proven, a visual aphasia, in which the abscess does not involve the sensory centre for speech itself, but the tracts which lead to it. Alexia and agraphia may also be often present.

If the abscess lies deeply in the temporal lobe, it may directly or through alterations in its vicinity produce symptoms which indicate a lesion of the *motor, optic,* and *sensory* conducting tracts.

Hemiparesis of the opposite side, generally accompanied by rigidity or contracture, occurs most often. Convulsions may also be found on the opposite side of the body. Hemianesthesia and hemianopsia are not uncommon symptoms. In a case of abscess of the right temporal lobe, observed by Jansen and myself, and operated upon by him, hemiparesis and hemihypesthesia, hemianopsia bilateralis sinistra, and conjugate deviation of the head and eyes towards the right were present.

The basal cranial nerves are often involved by an abscess of the temporal lobe, particularly the third, and occasionally the sixth, producing appropriate paralytic symptoms (particularly ptosis).

Abscesses of the inferior parietal lobe, as also those of the occipital lobe, may induce hemianopsia.

Blindness was noticed in a bilateral metastatic abscess of the left occipital lobe (Heinersdorf).

Purulent foci of the motor zone, mostly of traumatic, at times of metastatic, origin,—the embolic material passing particularly often from the lungs to the artery of the fossa of Sylvius,—produce the well-known phenomena of cortical epilepsy and paralysis (monoplegia and hemiplegia). In many cases the cortical paralysis develops step-like, the individual paralytic attacks being preceded by convulsions. This is probably due to the gradual purulent disintegration of the cortical matter. Larger abscesses of the cerebrum involve the motor conducting tracts more often than the sensory, so that hemiparesis is the most common, but also the most indefinite focal symptom. Motor aphasia has been occasionally noticed in abscesses of the left frontal lobe.

In *cerebellar abscesses* the pain is mostly in the occipital and nuchal regions; a slight cervical rigidity is also often present. Focal symptoms are often entirely lacking. *Severe vertigo* and *disturbance of coordination* are to some extent characteristic, but are only well marked in a few cases, and were even absent in a case with disintegration of the vermis; on the other hand, they were present in abscesses of other cerebral regions.

Bilateral amaurosis has been noticed several times in cases of cerebellar abscess. Pressure upon the pons, quadrigemina, and medulla oblongata, and the cranial nerves arising here, may cause nystagmus, paralysis of the ocular muscles, etc. The knee-reflex was absent in several cases of cerebellar abscess (Macewen).

Abscesses of the *pons* and *medulla oblongata*, which are very rare, betray themselves, if large, by the characteristic symptoms of bulbar disease. In a case cited by Eisenlohr, in which the focus lay in the lower part of the oblongata, all signs of bulbar paralysis of the cranial nerves were absent, but paraplegia of all four extremities was present.

If the abscess breaks through into the ventricles, very alarming symptoms occur,—generalized convulsions, delirium, passing rapidly to deep sopor, tetanic contractions, chills, high fever, etc. The previously slow pulse becomes rapid and irregular; the respiration rapid, or of a Cheyne-Stokes type. In this condition death comes on within a few hours. In other cases the patient dies from a rapidly spreading universal purulent meningitis.

Diagnosis.—This cerebral disease is not always easily recognized. The etiology, the detection of some moment which could produce cerebral abscess, is very important. If a trauma has occurred and is immediately or shortly followed by symptoms of cerebral disease, differentiation between abscess and hemorrhage is easily made, but purulent meningitis is very difficult to exclude. If the symptoms only occur after a period of latency, only *abscess* and *cerebral tumor* are in question. Fever, chills, a rapid progression of the disease from the time it becomes evident, and absent or late development of optic neuritis, in doubtful cases indicate an abscess.

A tumor generally develops insidiously and the symptoms gradually increase; focal symptoms are noticed earlier and choked disk is almost always present, and often prominent early in the course of the affection. Fever is generally absent.

The *traumatic neuroses* may also be confused with cerebral abscesses of traumatic origin. When the psychic disorder is of a hypochondriac, melancholic, or hysteric character, and the symptoms are present on the side of the body corresponding to the trauma, a differentiation is not difficult. It is otherwise in cases in which, after some cranial injury, vertigo, heaviness of the head, stupor, etc., are complained of, and the objective examination is negative or reveals only symptoms of doubtful value; as pupillary differences, acceleration of the pulse, etc. It should be the rule in such cases only to make a diagnosis of cerebral abscess when the symptoms are clear; fever, slowed pulse, cerebral vomiting, on the one side, focal symptoms on the other, reveal in doubtful cases the organic disease. The functional neuroses are characterized mostly by sensory disorders of a definite character, by vasomotor phenomena, etc. Attacks of head-pain, vertigo, stupor, vasomotor disturbances, fever, etc., may also follow minute alterations of the vascular system of the brain due to cranial injuries (Friedmann), but distinct focal symptoms corresponding to the seat of the injury are absent.

Otitic cerebral abscess is not always immediately recognized, as, especially in the young, a number of its phenomena occur in uncomplicated otitis, and particularly in exacerbations of the chronic process and retention of pus; to these belong headache, vomiting, vertigo, stupor, fever, even pupillary differences. Some observations even show that optic neuritis and ptosis may be due to this cause. All these phenomena have been made to disappear,—for example, by paracentesis of the tympanic membrane. Whenever these symptoms appear in the course of an aural disease, a local cause, as retention of pus, must first be thought of. If they persist after evacuation of the pus, after appropriate treatment of the aural disease, or new symptoms come on not explainable by the local process, the diagnosis of cerebral abscess gains in certainty. The appearance of optic neuritis in diseases confined to the ear is so uncommon that it should raise a suspicion, if present, of an intracerebral disease.

Labyrinthine disease and Ménière's vertigo should rarely be mistaken for cerebral abscess, though in one case they simulated cerebellar abscess. The combination of headache with vertigo, vomiting, and fever, also with nystagmus, may be due to simple labyrinthine disease.

External purulent pachymeningitis or *extradural abscess*, the most frequent complication of aural suppuration, may cause almost all the symptoms of cerebral abscess; the symptoms of intracranial pressure are, as a rule, less marked and the focal symptoms are more often lacking,—hemianopsia being always absent. The most important differential points are, however, the *local* signs of this disease,—swelling and pain behind the mastoid process, the fistular formation in many cases, the limitation of movements of the head, the *caput obstipum*, etc. Practically, the differential diagnosis is not of so much importance, as an extradural focus of pus is always sought after in doubtful cases, and has been discovered by the radical operation undertaken for the aural disease.

Otitic sinus thrombosis produces, as a rule, high remittent fever, increasing quickly and falling rapidly, chills, abundant perspiration, pyemic symptoms, metastases, etc., and, in addition, the objective signs of sinus thrombosis are present (compare with the next chapter). Atypical cases, resembling abscess, however, occur, and *vice versa*.

The greatest difficulty lies in distinguishing abscess from *diffuse purulent meningitis*. In typical cases, its symptomatology is very dissimilar to that of abscess; the acute development, rapid course, high fever, acceleration of the pulse (retardation of the pulse does not last long or reach a high degree), meningeal symptoms of irritation,—hyperesthesia of the skin and senses, psychic and motor restlessness, convulsions and temporary twitchings, muscular rigidity, spinal symptoms, etc.,—indicating clearly the disease. But in some cases of an atypical course the clinical picture resembles that of abscess, and it may be impossible from the symptoms to determine whether abscess is combined with meningitis or not. Lumbar puncture (see page 478), however, in most cases enables one to make a diagnosis (Lichtheim, Lentert). *Retarded pulse*, a temperature not far from normal, simple progressive stupor, and all phenomena which indicate a circumscribed seat of the disease (temporal lobe or cerebellum) denote an abscess.

Localized *purulent meningo-encephalitis* in some cases, particularly when it has established itself over the temporal lobes or cerebellum, may resemble cerebral abscess. For the differentiation of abscess from meningeal tuberculosis, see the description of the latter disease.

Serous meningitis, not a rare complication of purulent otitis, may simulate cerebral abscess on the one hand, purulent meningitis on the other. But it does not, as a rule, cause any focal symptoms (bilateral paresis of the extremities and cranial nerves and cerebellar ataxia have been noticed several times) or any marked or continuous rise in temperature, while choked disk and visual disorder are observed early and often, and, what is very important, it may spontaneously disappear or recede after evacuation of the pus in the ear or in the extradural region, or after opening up of the arachnoid space. Lumbar puncture may give evidence of this complication.

Acute non-purulent encephalitis may also follow an aural disease, but it generally comes on after an infectious fever, lacks the characteristic topical relationship with the aural trouble, and has a characteristic development and course (see page 525).

I have often noticed a *reflex neurosis* in aural diseases, which needs to be differentiated. The subjective disorders were sometimes very similar to those of abscess, but paresis and anesthesia upon the *side corresponding to the diseased ear* were almost always present, and these symptoms were sufficient data for a positive diagnosis. A girl whom I treated not long ago for this disease referred all her symptoms to the side corresponding to the diseased ear. She gave herself no peace : even in her sleep she scratched this side continuously until the blood ran.

The *local diagnosis* of otitic abscesses, particularly their location in the temporal lobes or in the cerebellum, is rarely made with certainty. Close observation of the ear, the seat of carious processes, the localization of pain and sensitiveness to percussion permit us to draw certain conclusions. But the focal symptoms given above enable us most clearly to determine this.

In general, abscess of the temporal lobe—especially of the left side—can be more positively diagnosed than one of the cerebellum. In the successfully operated cases of cerebellar abscess,—according to Koch, nineteen,—it was not so much the nervous symptoms which led to a local diagnosis as it was the etiologic factors (wearing away of the anterior wall of the posterior cranial fossa, dural fistula, etc.), or the cerebellum had been only explored after pus was sought for in vain in the temporal lobe.

Prognosis.—Cerebral abscess generally runs a lethal course if artificial aid is not furnished. It may make for itself a path to the tympanic cavity, oral or nasal orifice, or break through externally; but this happens rarely, and spontaneous recovery seldom occurs in this way. The disease then generally results fatally, either from cerebral edema and hydrocephalus, or from perforation into the meninges or ventricles. The patients, as a rule, die in deep coma. As soon as the disease has reached the terminal stage, the fate of the patient is sealed in case the abscess still remains. In recent years the prognosis has been somewhat better, since traumatic abscesses of late formation and otitic ones have been treated surgically. In sixty cases of cerebral abscess of traumatic etiology, which were operated upon, thirty-eight recovered; of one hundred and thirty-eight otitic cases, sixty-two were cured.

The results of the different operators vary considerably. Macewen was the most successful; he operated in nineteen cases of cerebral abscess and brought eighteen to recovery, one of which even being complicated with purulent meningitis. Schede, Schwartze, Gussenbauer, Esmarch, Bergmann, Starr, and others have reported cures. The figures given above do not include all the cases reported.

Abscesses of the temporal lobe offer, on the whole, better chances for recovery by surgical treatment than do those of the cerebellum.

Treatment.—*Prophylactic* measures are very important. Cranial injuries must be protected from infection, an infectious nidus in the neighborhood of the brain must be made innocuous and the brain guarded from infection. The treatment of cranial wounds, of otitis, of retention of pus in the temporal bone, etc., are treated of in text-books on otiatrics and surgery.

Bergmann particularly warns against forcible injections and irrigations of the ear.

In every case where the diagnosis of traumatic cerebral abscess can be definitely made, and in which a fatal complication can be excluded, evacuation of the pus is demanded. (Bergmann regards red softening of traumatic origin also as being operable.)

In every case where the diagnosis of uncomplicated otitic cerebral abscess can be definitely made, and where evident signs of breaking through, or of another disease, of itself fatal, are not present, surgical treatment is proper. First see if removal of the pus in the ear or in the extradural region will relieve the cerebral symptoms. Where this does not succeed, or the result is a negative one, the abscess in the brain should be sought after and evacuated.

In some cases, however, when a definite local and general diagnosis can be made, the purulent foci in the brain can be directly operated upon.

External purulent pachymeningitis, sinus thrombosis, and even beginning pyemia are not contra-indications to operative treatment; nor is circumscribed purulent meningitis.

If a diffuse purulent meningitis is present, it is advisable to abstain from operation. Some authors, however, do not think so. Lumbar puncture will generally tell us whether a diffuse purulent meningitis is present in addition to the cerebral abscess.

Metastatic abscesses cannot be treated surgically, as they are almost always multiple, and the cause is often incurable. A few good results have, however, been secured here also.

THROMBOSIS OF THE INTRACRANIAL SINUSES.

Anatomic Relations.—The transverse sinuses which traverse the transverse sulcus pass, together with the inferior petrosal sinus, into the internal jugular vein. In addition to the sinuses passing to the torcular Herophili, the external cranial nerves pour their blood into the transverse sinus, through a branch passing through the mastoid foramen,—a branch of the occipital and posterior auricular veins, which unite and form the external jugular vein,—and also the cervical veins through a branch which pass through the posterior condyloid foramen and join the transverse sinus and the cervical plexus.

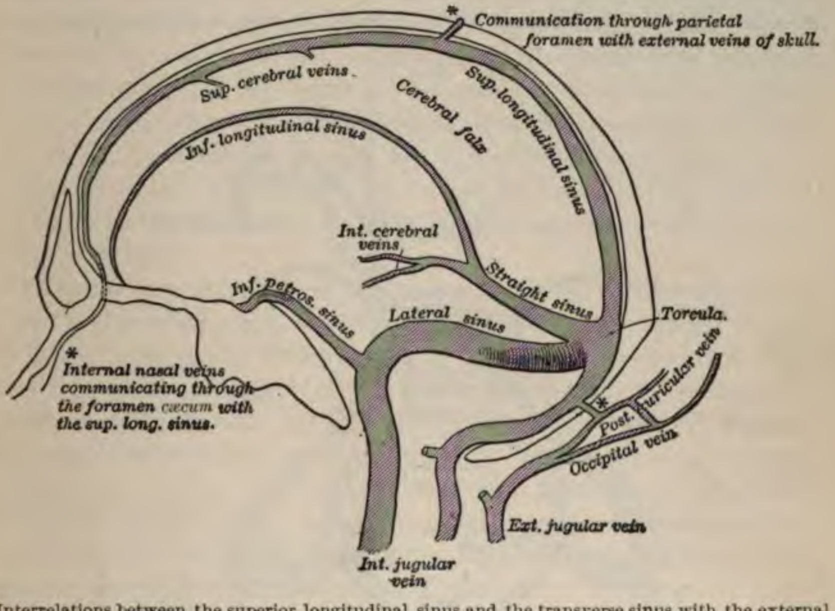
The superior longitudinal sinus passes from the foramen cæcum of the frontal bone, through which it connects with the internal nasal veins, to the internal occipital protuberance. The external cranial veins are connected with this sinus through the parietal foramen. The cavernous sinus is joined at its anterior end by the opthalmic veins, whose upper branch connects with the anterior facial vein at the inner canthus of the eye.

Symptomatology.—Sinus thrombosis is rarely a purely uncomplicated disease. Its symptomatology is commingled with other diseases, and must be extracted from a symptom-complex made up of a combination of different abnormal conditions.

There is a *primary* and a *secondary* sinus thrombosis. The latter is the result of a *phlebitis* of the sinus wall, or one of the veins entering into it, and this again is the result of an inflammatory (mostly septic) *process in the neighborhood.* Secondary thromboses are also mostly of infectious origin.

Primary sinus thrombosis is, as a rule, a result of *cardiac weakness;* it is, therefore, called *marasmic.* It is found particularly in infants and in the aged. In children, it is generally due to an exhausting diarrhea. The cardiac weakness produces a retardation of the blood-current, which in the sinus easily causes a coagulation, as the circulation here is very poor. Thickening of the blood and a decreased quantity of blood may favor the coagulation, especially as the inelastic sinus wall does not accommodate itself to the quantity of blood, and the trabeculæ running through the sinus also aid in the formation of a coagulum.

FIG. 236.



Interrelations between the superior longitudinal sinus and the transverse sinus with the external veins. (*) (After Leube.)

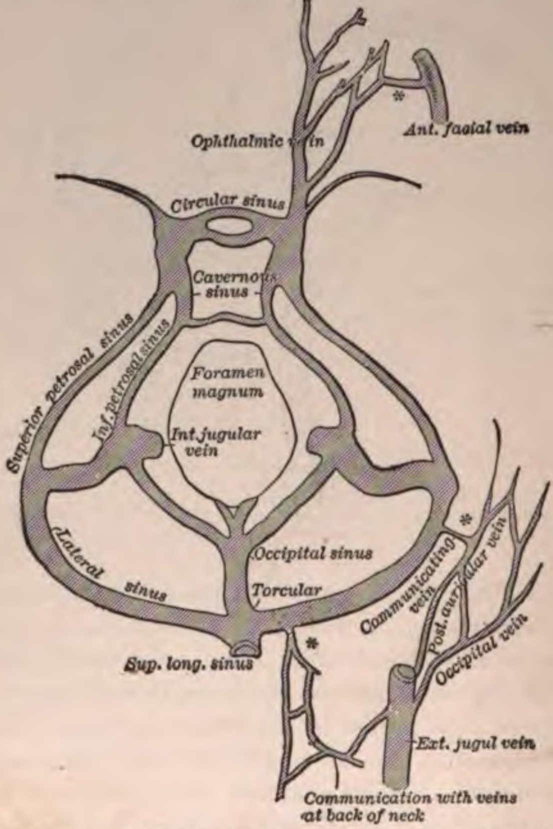
The disease may develop in the course or in the terminal stage of *exhausting diseases*,—*e.g.*, in tuberculosis, carcinoma, etc.; more rarely in the course of the acute infectious diseases,—*e.g.*, typhoid, pneumonia, variola, etc. The fact established by Bollinger, that *chlorosis* often produces sinus thrombosis, is of particular importance. Andrew, Church, Proby, Pasteur, Göbel, Nonne, Kockel, and others have made similar observations. In addition to cardiac weakness, which is probably also in play here, *fatty degeneration of the sinus endothelium* is blamed, also increase in the number of blood-plates. Simple anemia, on the contrary, produces only rarely thrombosis of the cerebral sinuses; it has been observed after repeated losses of blood.

Secondary phlebitic sinus thrombosis is mostly a result of diseases of the bones lying near the sinuses or communicating with them by veins. Of the thromboses of this origin the *otitic* take a very prominent place.

Otitis media, caries of the petrous bone, and cholesteatoma are frequent causes of sinus thrombosis. The carious process attacks the sinus wall directly, or a *thrombophlebitis* of a vein, connecting the internal ear with the sinus, extends to it. Just in these cases are complications often found,—*meningitis*, *extradural* or *cerebral abscess*. *Pyemia* is a result of the septic character of the thrombosis, in that the material reaches the jugular vein and from here gains access to the system.

Caries of other cranial bones, purulent and tubercular meningitis, also purulent septic diseases of the cranium, face, and its orifices may produce sinus thrombosis through the emissaria Santorini, for almost every sinus receives veins from the external aspect of the cranium. Wounds, carbuncles of the face or neck, parotitis, facial erysipelas, orbital plegmon, panophthalmitis, etc., may cause sinus thrombosis. It may also be an accessory phenomenon of general septicopyemia.

FIG. 237.



Interrelations of the transverse and cavernous sinus with the external veins. (*) (After Leube.)

It may also be of *traumatic* origin, and be produced by compression of the sinus.

Heubner has recently discovered, in two cases of apparently primary (spontaneous) sinus thrombosis, micro-organisms in the thrombus (and in the cerebrospinal liquor).

Pathologic Anatomy.—The thrombosis may be confined to one or a part of one sinus, or extend to several, even to all of the sinuses. We find in the diseased sinus a recent grayish-red or an older, hard, white thrombus adherent to the walls. The older the thrombus is the closer it adheres to the wall. In cases of secondary thrombosis it is generally decolorized, puriform, and sanious; the wall of the sinus also appears altered, being greenish or greenish-yellow, etc. Masses of pus are found not only in the sinus, but also in the jugular, subclavian, and even in the superior cava. Abscesses on the wall of the sinus are often observed in cases of phlebitic thrombosis. The thrombi extend generally into the tributary veins.

Thrombi may extend from the perpendicular sinus into the magna Galeni and internal cerebral veins, and induce hydrocephalus, though this is not a constant or even frequent result of sinus thrombosis, as some authors assume. The part of the veins not thrombotic is tortuous, dilated, and filled with blood. The corresponding cerebral regions, from which these veins carry the blood, are hyperemic, with capillary hemorrhages, infarcts, foci of softening, and, more rarely, abscesses. Meningeal hemorrhages are more rare. The complications of secondary thrombosis have already been described. I have found very often in the marantic form an abnormal smallness of the heart.

The *seat* of the thrombosis varies according to the *cause*. The marantic occur almost always in the longitudinal sinuses, especially their posterior parts, and probably also in the transverse sinuses; the otitic, mostly in the transverse sinus (sigmoid fossa), at times in the cavernous and inferior and superior petrosal. They often extend from one sinus into another, even into those of the opposite side, and not rarely involve the (bulb of the) jugular vein. The traumatic attack particularly the region of the superficial sinuses; those arising from inflammatory processes in the face, eyeball, etc., the cavernous sinus.

Symptomatology.—Cases of marantic thrombosis are generally obscured by the basic disease and by symptoms of cerebral anemia; cases of secondary thrombosis, by caries and by complications (meningitis, abscess). The symptoms which are the most characteristic of this disease—those which are the direct result of obstruction of the sinus—are least marked in most cases. These are *symptoms of congestion* in the external *cranial* and *facial veins*, which are directly connected with the sinus or the internal veins which enter it. Thrombosis of the cavernous sinus, for instance, produces dilatation of the *frontal veins*, cyanosis of the orbital and frontal regions, swelling of the eyelids and of the neighboring regions, *protrusion* of the *eyeball* from dilatation of the retrobulbar veins, in a few cases *congestive symptoms in the retinal veins*, and even *choked disk* has been observed, though Jansen did not notice these phenomena in his cases. Amblyopia and amaurosis have often occurred; and in one of my cases with a normal ophthalmoscopic picture. Thrombosis of the central retinal vein has been described. In many cases, *neuralgic pains* in the first trigeminal branch and paralytic symptoms in the *abducens*, *oculomotor*, and trochlear which pass along the wall of the sinus belonged to the clinical picture, though, according to Jansen, more rarely than some imagined.

Thrombosis of the *transverse sinus* produces in many cases an *edematous swelling* of the soft tissues behind the mastoid process (Griesinger) and a dilatation of the veins of the skin. A continuation of the thrombus into the jugular may often be detected by palpation; Jansen found the jugular involved in about one-third of the cases of otitic thrombosis of this sinus. Sensitiveness of the jugular vein either spontaneously or from pressure, feeling of a hard sensitive cord, swelling of the soft structures and of the glands in the neighborhood, pain in the side of the neck on moving the head, torticollis, and pain in deglutition are symptoms observed.

Lymphangitis and gravity abscess may simulate a phlebitis of the jugular. Gerhardt has called attention to the lesser fulness of the external jugular vein in comparison with that of the sound side (because it gives up its blood more easily to the less-filled internal jugular vein). Jansen could not detect this, and it has generally been found lacking. Paralytic symptoms of the glossopharyngeal, vagus, accessory, and hypoglossus have also been described, but they occur very rarely.

In thrombosis of the *longitudinal* sinus, congestion and torsion of the veins of the frontal, parietal, and temporal region with edéma were noticed in some cases. Bleeding from the nose has also been frequently observed, and has been thought to be due to overfilling of the nasal veins. In children, the fontanelles are tense and bulging on account of the congestion of the cranial veins and the increased intracranial pressure.

These diagnostically valuable signs are very inconstant; the common phenomena of sinus thrombosis are of rather an *indefinite* nature.

They are in many respects similar to those of meningitis and of focal diseases, as softening and hemorrhage into the cerebral substance are almost regular, and *abscess* not an infrequent complication of this disease.

In *autochthonous* cases of sinus thrombosis the following phenomena have been observed. The patient became sick suddenly—or after chlorosis, hydrocephaloid disease, phthisis, etc.—with severe *headache; vomiting* and *stupor* came on, the latter increasing to *sopor; delirium*, generalized or unilateral *convulsions*, and *paralyses*, and, more rarely, disorders of co-ordination, contracture, tremor, etc., occurred. In many cases rigidity of the back of the neck and also of the extremities was present. The paralysis involved the extremities of one side of the body, mōre rarely the cranial nerves, particularly the ocular nerves. Aphasia and hemianesthesia were infrequent symptoms. The *temperature* was normal or elevated, an increase in leaps was observed many times, and on several occasions an *increase* to over 108° F. The pulse varied, being mostly normal or retarded in the beginning, generally accelerated near the end; the same was true of the respiration. The patients generally died in a few days, in some cases after one or two weeks, in a comatose condition.

From this description it can be seen that a diagnosis must be based upon the etiology and the symptoms of congestion which may be present, as the general symptoms arouse a suspicion of meningitis or of some focal disease. The latter are also really present,—*i.e.*, hemorrhagic and softened foci in the hemispheres. They were absent in a case described by Nonne, though in it the cortical epilepsy, hemianesthesia, and hemianopsia indicated an extensive focal disease. If symptoms of congestion, etc., are absent, no positive diagnosis can be made, and confusion with meningitis, cerebral hemorrhage, encephalitis, etc., is possible and excusable. The symptoms of a severe cerebral disease may also occur in the course of chlorosis, its anatomic basis being still unrecognized (Immermann, Eichhorst, Müller, Burton-Fanny, and others). In infantile hydrocephaloid disease, the exhaustion and cerebral anemia alone may induce a similar symptom-complex.

Secondary phlebitic, particularly otitic, sinus thrombosis is more easily diagnosed. Uncomplicated thrombosis of the transverse sinus caused by middle ear suppuration, when not septic, and excluded from the blood-stream by solid thrombi, need not, outside of fever and headache, cause any symptoms (Jansen). As a rule, it gives rise to symptoms which partly occur in the same manner in meningitis, cerebral abscess, and extradural suppuration, but which are partly characteristic, even pathognomonic. An extradural, perisinuosal abscess is often the transmitting factor, so that its symptoms, being ordinarily the premonition of thrombosis, should be carefully noted. They at least indicate a threatening danger. Jansen includes as symptoms of these abscesses, inflammation of the bones, subperiosteal abscesses, and phlegmons behind the mastoid process, especially around the mastoid foramen and neighboring part of the occiput, also upon the posterior part of the mastoid process itself, pain on pressure and percussion in this region, limitation of the movements of the head, especially on its sagittal axis, caput obstipum, nystagmus, etc. Then follows a series of phenomena which may be produced by the extradural suppuration as well as by the thrombosis,—optic neuritis and choked disk, which occur more often in sinus thrombosis (thirty-four to forty-five per cent. of all cases) than in extradural abscess, also headache, vertigo, vomiting, retarded pulse, irritability, sensitiveness of the spinous processes to pressure, rigidity of the back of the neck, and other meningeal symptoms of irritation. The diagnosis becomes certain when symptoms of pyemia or of jugular phlebitis and periphlebitis appear. Pyemic fever,—remittent, with abrupt rise to 104° to 108° F. and fall far below normal,—acceleration and irregularity of the pulse, repeated chills, profuse perspiration, diarrhea, icterus, swelling of the spleen, metastases, especially pulmonary ones (pulmonary infarct, pulmonary abscess), more rarely metastatic abscesses of the joints, liver, etc., are all characteristic symptoms and indicate that the process has extended to the sinus and that septic material has invaded this region. Hessler particularly has given a full description of this pyemia of otogenous origin.

Local external symptoms of sinus thrombosis are also characteristic, of which those with involvement of the jugular vein, which produce phlebitis and thrombosis of it, are particularly important.

Foci of pus may also develop in the vicinity of the mastoid and condyloid veins, more rarely in that of the deep veins of the back of the neck. We occasionally observe symptoms of compression of the glossopharyngeal, vagus, and accessory. A retardation of the pulse, which otherwise is not observed with sinus thrombosis, may then occur.

Spinal symptoms, such as flexion contracture of the lower extremities, paraparesis, and Westphal's symptom, have been several times observed in sinus thrombosis, which in such cases is probably combined with serous meningitis. Otitic sinus thrombosis may, particularly in children, resemble meningitis, even where it is not complicated by it. Pyemic symptoms may represent the clinical picture, while all local signs are missing. Finally, it should be remembered that thrombosis is very often combined with purulent meningitis and cerebral abscess. Lumbar puncture may tell us eventually whether a purulent meningitis is present in addition to the thrombosis (Lentert).

The thrombosis may extend from the transverse sinus to the inferior petrosal and *cavernous* sinuses. The latter may also be directly affected, and here it is the rule that it springs from the cavernous sinus of one side to that of the other (Macewen).

The prognosis of sinus thrombosis is unfavorable, though we are not justified in considering it an incurable disease. Spontaneous recovery occasionally occurs in primary thrombosis, probably from reabsorption of the thrombus, but circulatory disorders are not even a necessary result of a chronic thrombus. Recovery with persisting focal symptoms has also been noticed.

Recovery in phlebitic thrombosis is more rare, though observations of pyemia cured after sinus thrombosis have been made.

In infectious thrombosis the chief dangers are pyemia and purulent meningitis.

The danger of pyemia is greater in thromboses of the transverse and inferior petrosal than in those of the cavernous and superior petrosal sinuses. If the thrombosis extends from the transverse sinus to the torcular Herophili, meningitis almost always develops (Jansen). The prognosis of secondary thrombosis has been materially aided by *operative* treatment. There is no chance for recovery in cases complicated by diffuse purulent leptomeningitis, and but a slight chance with circumscribed purulent leptomeningitis, cerebral abscess, extensive pyemic metastases in the lungs, etc., though operation in some cases with beginning pyemia has given good results.

Treatment.—In cases of marantic sinus thrombosis, the treatment is symptomatic. The circulation should be aided, the cardiac strength increased by stimulants, nourishing diet, etc. Bloodletting is contraindicated in all cases.

The headache is combated by the use of an ice-bag and by narcotics. The patient should assume a recumbent position, with head slightly elevated.

In cases of phlebitic thrombosis, the pus must be evacuated. The more completely the pus is removed from the vicinity of the sinus the more caution must be taken to guard the thrombus from septic disintegration. Prophylaxis is here of much importance (incision of a carbuncle, of the infiltrated parotid, *trephining of the mastoid process*, *evacuation of extradural abscesses*, etc.). In recent years operative measures have been successfully used. Zaufal opened the way in 1880, then came Lane, Ballance, Parker, Salzer, but particularly Macewen and Jansen. Macewen cured twenty in twenty-seven operated cases; Jansen, eleven in twenty-four. Henschen grouped the figures of Macewen, Chipault, Jansen, and Koerner, and found that of one hundred and forty-five cases, eighty-five, or fifty-eight per cent., were cured. Opinions differ as to whether the internal jugular should be previously ligated or not, to prevent pyemia. Zaufal, Koerner, etc., are in favor of it, while Jansen does not consider it forbidden in a thrombosis confined to the sinus.

Thrombosis of the longitudinal sinus is seldom operable. The danger of air-emboli is slight and rarely observed (Kuhn).

CEREBRAL TUMOR.

The brain is a favorite seat for neoplasms. There is hardly a form of tumor which spares the cerebrum. The most common forms occurring are *glioma, solitary tubercle, sarcoma,* and *gumma. Carcinoma* is more rare, and *fibroma, lipoma,* and *osteoma* very uncommon. Psammoma and cholesteatoma, cavernous angioma, and dermoid cysts are of infrequent occurrence and of no practical importance.

Mixed forms of glioma and sarcoma are not uncommon; glioma may assume a myxomatous character. Osteosarcoma, fibrosarcoma, and angiosarcoma are forms occasionally found in the brain.

The most frequent cerebral tumors of adults are glioma, syphiloma, and sarcoma; tubercle is most frequent in childhood. All these forms may, however, occur at any age, though tubercle is rarely observed after the thirtieth year.

Gliomata vary from the size of a hazel-nut to that of a hen's egg, or even to the size of a fist. The borders are not sharply defined, do not extend by *compression* but by *infiltration* of the cerebral matter, so that it cannot, as a rule, be shelled out. A cross-section appears yellowish white to reddish gray, the color being often not very different from that of the normal cortical or medullary substance. If extensive hemorrhages have occurred in the tumor, the character of the tumor may be so obscured that hemorrhagic softening is diagnosed by the inexperienced. Closer examination will always reveal, particularly in the peripheral parts, islands of neoplasmic tissue. Cystic degeneration may also cause confusion; here also a mantle of tumor tissue is found in the periphery. Cavities covered with cells rarely occur in gliomata. The process may also resemble a local hypertrophy. Glioma does not involve the meninges or the bones.

Sarcomata arise frequently from the meninges, periosteum, and bones, and often only compress and disturb the neighboring parts of the brain. They frequently, however, grow into the cerebral substance or commence in the brain-tissue; even then, however, they are sharply defined and are separated from the normal brain-tissue by a zone of softening. Sarcomata are of various sizes, from that of a nut to larger than the fist. Fibrosarcomata originating from the periosteum become particularly large. On the whole, sarcomata are of a harder consistency than gliomata. Caseation and disintegration of some parts of the tumor from fatty metamorphosis is occasionally observed. It may be impossible to distinguish glioma from sarcoma with the naked eye. A diffuse sarcomatous condition of the meninges may be present, in addition to the tumor.

Carcinoma is a soft, vascular tumor of irregular outline, which may spread itself superficially over the dura, or form a circumscribed or diffuse tumor in the inner part of the brain. Wernicke claims that they may reach the size of a child's head, and emphasizes the pronounced tendency to pultaceous softening and to disturbance of all the tissues.

Syphiloma and *solitary tubercle* resemble each other very much at first glance. They are generally small tumors of the size of a hazel-nut or walnut, though a tubercle may approach the size of a goose-egg. Both are non-vascular, and have a tendency to caseation; tubercle, in addition, tends to purulent disintegration. The more recent peripheral parts of the neoplasm have the characteristics of granulation tissue; an eruption of miliary tubercles in this or neighboring parts reveals the solitary tubercle. A further differential sign is that gummata almost always arise in the *meninges*, and the connection between them can still be distinctly recognized; their tendency to extend along the surface is also noteworthy. The most exact criterion for the differentiation is, however, a bacteriologic examination; *tubercle bacilli* are generally found in the solitary tubercle.

Psammoma, which occurs almost exclusively in the meninges and pineal gland, is a neoplasm richly infiltrated with lime-salts, and which frequently possesses no tendency to grow. *Adenoma* form in the *hypophysis*.

Cholesteatomata, which establish themselves almost entirely at the base of the brain, especially near the petrous bone, are, as a rule, an occasional post-mortem find. The name "pearly tumor" is based upon the formation of white, shining nodules, which give the tumor an appearance similar to mother-of-pearl.

Cysts of the brain are either of parasitic origin (cysticerci and echinococci) or degenerated neoplasms. (Cysts due to hemorrhage and softening are not included here.)

Localization of the Tumors.—They may have their seat anywhere, though all regions are not involved with equal frequency. Most tumors develop in the cerebrum, particularly in the medullary substance. The cerebellum is next in frequency; then come the pons and the central ganglia, following which, the quadrigemina, etc. Glioma occurs particularly in the hemispheres of the cerebrum, also of the cerebellum; rarely anywhere else. Solitary tubercle favors the pons, cerebellum, and cerebral cortex. Syphiloma is rare in the cerebellum, still rarer in the central ganglia. Sarcomata grow particularly from the periphery into the cranial cavity; the bones of the base of the skull, temporal, and sphenoid bones, less so the occipital, are often its places of origin.

Other Peculiarities (Rapidity of Growth, Multiplicity, etc.).—Glioma develops slowly, often coming to a complete stand-still. A rapid swelling is found only when a large exudate of blood occurs into the substance of the tumor. The softer sarcomata enlarge more rapidly than the hard ones; the osteofibrosarcomata are particularly of slow growth. Carcinoma proliferates rapidly. Tubercle may enlarge rapidly; it may, however, remain stationary for a long time, and undergo retrogressive alterations; sudden proliferation and regressive metamorphosis is particularly characteristic of syphiloma.

Gliomata generally occur singly, as do sarcomata. The former is always primary; the latter is sometimes of metastatic origin. Carcinomata, which are mostly, perhaps always, of a metastatic character (primary tumor in mammæ, lungs, pleura, etc.), are rarely found singly. Multiplicity is also the rule for syphiloma and tubercle. In one hundred out of one hundred and eighty-five cases, several tubercles were found in the brain.

What influence has the development of a tumor in the brain upon the entire organ and its vicinity? We can generally detect, after the cranium is opened, that a neoplasm has attacked the brain. The gyri are mostly flattened; the sulci are smoothed over; it is seen that pressure from within has arched out the cortex. The pia and the brain itself seem dry, generally also anemic. These alterations involve particularly that section of the brain in which the tumor is found; they are absent when it arises from the cortex or from the membranes of the brain. The part of the brain around the tumor is generally softened. The parts of the brain under pressure from the tumor may show various displacements and alterations. In a case of cerebellar tumor, for instance, observed by me (Fig. 238), the corresponding half of the pons was squeezed into one-quarter of its volume.

The cerebrospinal liquor is almost always increased; *internal hydrocephalus* is particularly pronounced in tumors of the cerebellum and quadrigemina, as the tumor by pressure displaces the communication between the ventricles, or venous congestion and increased transudation comes on as a result of compression of the veins (great Galenic) which carry the blood from the choroid plexus into the straight sinus.

As to the cause of the so-called choked disk, see page 448. The *nerves* at the base of the brain are often flattened either directly by the tumor or from the increased cranial pressure, and especially when a pronounced hydrocephalus accompanies the tumor. This flattening is noticed on the oculomotor, but more often on the abducens and olfactory nerves. The floor of the third ventricle may be pushed forward so much by the hydrocephalus as to compress the chiasm directly. The bony cranium itself undergoes alterations. *Osteoporosis* of the neighboring parts of the skull is a not infrequent concomitant of superficially located neoplasms, and in tumors lying deeply within the brain also; general increase in intracranial pressure may cause an osteoporosis extending over the entire cranium (particularly the roof). Perforation rarely occurs, if we exclude tumors of the periosteum and dura, which proliferate outward.

The increased intracranial pressure may produce in children a springing of the sutures which have already grown together.

Alterations in the spinal cord have recently been noticed in cerebral tumors, especially in the posterior tracts and roots (Dinkler, Mayer, Anton, Pick, Hoche, Ursin). They are probably an expression of the injury caused by the increased intracranial pressure. Toxic factors and nutritive disturbances may also be in play.

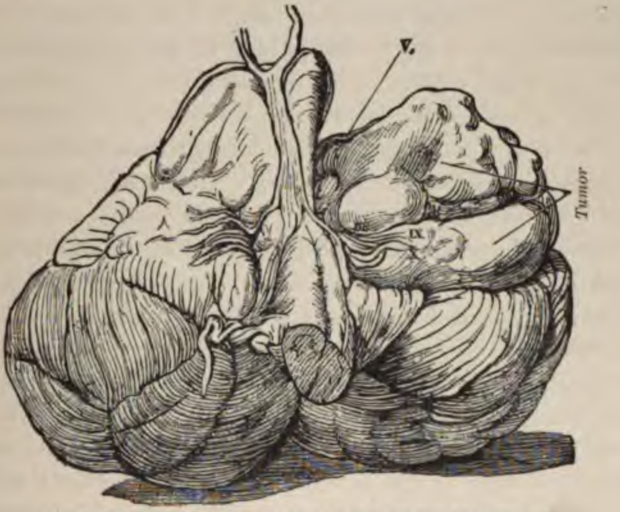
Symptomatology.—Brain tumors cause a series of symptoms which are independent of their seat,—*general cerebral symptoms*; others dependent upon a lesion of a definite part of the brain, varying with the seat of the neoplasm,—*focal symptoms.*

The first includes *choked disk, headache, stupor, vomiting, vertigo, retardation of the pulse, and generalized spasms.*

Of these symptoms the objective ones are the more important, and among these optic neuritis, or choked disk, is the most pathognomonic.

Choked disk is characteristic of tumor, being due to brain tumor in ninety out of one hundred cases, and being only lacking in ten to twenty per cent. of such cases. It is almost always bilateral, though it is often more pronounced in one eye than in the other. Optic neuritis is closely allied to—probably only a slighter grade of—choked disk. It often precedes the development of a true congestive papillitis, and is less pathognomonic in itself than is a true choked disk, as slight degrees of optic neuritis occur in the most varied disease processes (page 447).

FIG. 238.



Cerebellar tumor with atrophy of the pons due to pressure.

Headache is an almost constant symptom of cerebral tumor. It may be missing in the initial stage, or be slight or appear intermittently. Later on it is rarely lacking. It is intensive, becoming more severe than in any other disease, and in the later stages is constantly present; but it may exacerbate at times. It is increased by every forced expiratory act, as coughing, straining, and sneezing. It is not amenable to simple psychic treatment nor to the drugs which relieve a simple (nervous) headache. It is generally a diffuse pain extending over the whole head; it is sometimes found in definite areas,—for example, in the frontal or occipital region. To what extent its seat is of value for local diagnosis will be discussed later.

Stupor is a sign of much importance. It may be absent in the first stage, also when the tumor is very small, or when its seat is outside the cerebrum,—*i.e.*, extracerebral, on the base of the brain, cerebellum, etc. Otherwise we may regard it as a rule that the sensorium is not free at the height of the disease. The patient can answer questions asked of him, but it can be seen that it is difficult for him to do so. He resembles a person who is "drunk with sleep;" in the advanced stage continuous *lethargy* is present, so that the patient goes to sleep at the table, retains his food in his mouth, and also urinates and defecates on such occasions. Slighter grades may be noticed earlier.

Other psychic defects occur sometimes. Melancholia, hallucinatory excitement, simple dementia, finally a form of mental weakness associated with a peculiar exaltation are the conditions observed. We will refer again to this when we discuss the question of localization.

Vomiting is not a constant symptom, but is present in most cases. It is a true cerebral vomit, may be one of the initial symptoms, but generally only comes on long after the headache. It often occurs at the height of an attack of headache. In tumors of the posterior cranial fossa (cerebellum and oblongata) we find it particularly pronounced.

Vertigo.—A persistent dull feeling in the head similar to that in intoxication is often complained of and styled vertigo. Attacks of real rotatory vertigo, in which the patient loses his co-ordination and falls to the ground, or must hold to something to keep from falling, are rarer. It is particularly marked in cerebellar tumors.

Attacks of *unconsciousness* with *convulsions* are quite frequent in brain tumors. We do not mean attacks of cortical epilepsy, which only occur in tumors of certain areas.

We must include among the general symptoms in the sense defined above attacks similar to true epilepsy, which occur at all stages of the disease, and which may be prodromata. The epileptic attacks may precede the outbreak of the other symptoms by years, so that it is hardly possible to regard them as an initial symptom; it is more correct to look upon the epilepsy as an independent disease and to consider it possible that a brain so organized is particularly suitable for a neoplasm. I have seen cases of this kind in which the post-mortem revealed a partly bony-hard tumor, which had probably been developing for years.

In place of typical epilepsy *attacks* of *simple unconsciousness* alone or combined with *automatic* and *involuntary movements* occur, also simple convulsions with intact or impaired consciousness. The spasms may also resemble those of hysteria.

A tetanic rigidity of the musculature with retraction of the head, and with normal or slightly impaired consciousness, is often noticed in tumors of the posterior cranial fossa.

In a large number of cases the pulse is permanently or temporarily retarded (as low as forty-eight beats a minute, or even lower). This is never an early symptom. It is a sign of increased cranial pressure, and the other symptoms of this condition usually precede it. It may occur earlier in tumors which directly involve the vagus region. The irritation tends, however, to cause acceleration of the pulse, a phenomenon otherwise met with at the terminal period of cerebral tumor.

Focal Symptoms.—In some cases of brain tumor only the general symptoms or a part of them are present during the entire duration of the malady. We are able to make a diagnosis of cerebral tumor, but cannot determine its location. Small tumors may occur almost anywhere without inducing focal symptoms, particularly in the cerebral medulla where they force the fibres apart without impairing the conducting power. Large tumors in the right frontal, right temporal, and a part of the right parietal lobe are not accompanied by characteristic focal symptoms. These parts of the brain, it seems, may be injured or impaired without any pronounced symptoms indicating it; at least, we are not able to recognize any. There was a time—and it was not so long ago—when it was thought localization was impossible, due to the above-mentioned fact and to the more important one, that the tumors act in an irritative and paralyzing manner upon distant parts of the brain. This was called action from a distance. Its importance was overvalued. The pressure which a tumor exercises, though it influences the whole brain, acts much more strongly upon its immediate vicinity, upon the tissues on the side of the brain it is on, rather than upon those of the opposite side, etc. Focal symptoms, therefore, are not of much local diagnostic value if the symptoms of general cerebral pressure are very pronounced. Phenomena due to the action of the tumor upon the circumferential parts can be called neighborhood symptoms (Nachbarschaftsymptome).

Tumors of the Motor Zone.—These cause very pronounced focal symptoms, which precede the general cerebral phenomena by some time. Symptoms of cortical epilepsy are, as a rule, noticed first, a tonic muscular rigidity or convulsions which begin in the face, an extremity, or a muscular group, remain confined to this region awhile, and spread over one-half of the body in future attacks. Consciousness is intact, at least in the beginning (page 433). Paralytic symptoms come on later, at first of a transitory nature, appearing only after the attacks, but they gradually increase in intensity and extent, and finally become stabile. They are generally *monoplegic*, facial, brachial, crural, faciobrachial, etc., according to the seat of the tumor. The disassociated occurrence of paralysis is a particularly characteristic sign of neoplasms in the motor region. With the growth of the tumor the monoplegia may also extend and become a hemiplegia. An attack may be ushered in by paresthesia of one extremity or of the face, or this may be all that occurs; twitchings generally appear at the same time or come on soon after the paresthesia. Decreased sensation is often found objectively at the same place. Such attacks justify the assumption that the tumor lies in the cortex of the motor zonē or close by it (meninges, subcortical medullary substance). The onset of twitchings in a definite muscular area and, later, paralysis of this region show that the centre in question is the place of origin of the tumor.

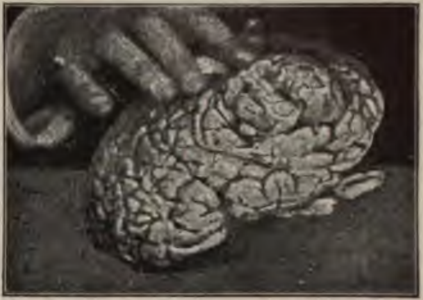
On account of the irregular extent of many tumors and the varying directions that they take in their growth, we cannot expect that these symptoms will develop in due regularity. A hemorrhage into a tumor may cause a sudden paralysis. The tumor may remain latent, and a hemorrhage into it or a circumferential softening may induce a paralysis without any symptoms of irritation preceding it.

The tumor may extend into the salient parts of the brain, merely impinging upon the motor region, and thus have a much larger circumference than is imagined. Tumors of the central ganglia may also induce motor irritative and destructive symptoms of this nature, but they almost always only develop in the later course of the disease. Glioma, sarcoma, syphiloma, tubercle, and cysticerci are particularly responsible for the above symptom-complex.

Vasomotor disorders and tachycardia have been noticed in but a few cases of tumors of the motor region.

Tumors of the Speech Centres.—The neoplasm not rarely has its seat in the left frontal or temporal lobes. If the third left frontal lobe is directly involved, motor aphasia occurs early, gradually increasing in degree. When once complete, it does not disappear. If the tumor lies in other parts of the frontal lobe, the speech defect may be absent continually or for a long time. As it grows, however, the speech in time becomes, as a rule, more and more affected. It seems to me that a sort of distaste for speech—a mutism—is often present, though the patient is still able to speak if necessary. But the words come out slowly; the answers are not only delayed, but a pronounced *bradylalia* is present. Tumors arising from the orbital surface of the left frontal lobe need not cause any aphasia. I saw one case, for instance, in which the tumor arose from the dura and penetrated deeply into the brain on the lower surface of the left frontal lobe, in which aphasia only occurred towards the end of life. Tumors of the left temporal lobe, particularly when they attack the first convolution, produce word-deafness, amnestic aphasia, and paraphasia. In a case which I observed not long ago, the tumor which arose in the supramarginal gyrus, lay in the posterior part of the fossa of Sylvius upon the first temporal convolution (Fig. 239) and extended somewhat into it. Aphasia only occurred when the patient stood upright.

FIG. 239.



Tumor of the left supramarginal gyrus, with slight involvement of the first temporal convolution.

Auditory hallucinations which occur in some cases are included among the focal symptoms of the temporal lobe. If the tumor lies deeply, it may cause hemianesthesia, hemianopsia, and hemiplegia from injury to the corresponding conducting tracts. A neoplasm which sits deeply in the medulla, in the *cornu ammonis*, and in the *central ganglia*, may, if large enough, induce aphasia, generally of a mixed type, and mostly only of a partial character, by extending to the speech centre or involving the tracts coming from it.

Tumors which affect the *left lower parietal* lobe directly or from pressure may cause alexia and agraphia.

Optic aphasia may be induced by tumors of the posterior basal part of the left temporal and neighboring parts of the occipital lobe (lingual gyrus). With left-handed persons, tumors of the same parts of the right hemisphere may produce the different forms of aphasia.

Aphasia is the only characteristic focal symptom of tumors of the *frontal lobe*, though we have a series of phenomena which are of some value in local diagnosis. It seems that a psychic disturbance (simple dementia or a morbid exaltation) occurs particularly early, and is very pronounced in such tumors. Tumors of the posterior part of the frontal lobe may, by involving the motor zone, cause spasms. Their onset with conjugate deviation reveal their frontal origin. Rigidity of the back of the neck and cerebellar ataxia also occur in neoplasms of the frontal lobe.

Tumors arising from the *parietal lobes* produce irritative and paralytic motor symptoms (probably by compression).

Sensory disorders, particularly of position, and ataxia are probably direct focal symptoms (see page 397). Alexia and visual aphasia may be symptoms of tumors of the left inferior parietal lobule. If the tumor is deep-seated, hemianopsia may be present.

Tumors of the Visual Region.—Tumors of the optic tract, pulvinar, and quadrigeminal region, as also those which break the continuity of the optic radiations on their way to the occipital lobes, and those of the occipital lobe itself cause *hemianopsia.* This would not have much localizing value did not the accompanying symptoms help to indicate the localization. Tumors of the optic tract involve, as a rule, other basal cranial nerves, particularly the oculomotor. Tumors of the thalamus generally produce an affection of the motor and sensory conducting path. If the gemina are involved, paralysis of the ocular muscles, etc., generally occurs. If the visual tract is involved on its way to the left occipital lobe, aphasia may also occur. If the tumor involves the occipital lobe, hemianopsia is the only focal symptom. In addition, unilateral facial hallucinations were observed several times. Bilateral, probably also unilateral, tumors of the occipital lobe may produce psychic blindness.

Tumors of the Central Ganglia.—The diagnosis is here very difficult, and is also not of much practical interest. Focal symptoms may be missing for a long time or permanently. As, however, the internal capsule is generally involved (from direct involvement or through pressure), *hemiplegia* is present, a hemiplegia which comes on gradually and is only partial. Indefinite unilateral twitchings, unlike those of cortical epilepsy, hemichorea, hemiathetosis, involuntary and automatic movements, are all noticed, also hemianesthesia and hemianopsia of the opposite side.

The more the tumor presses against the cortex, the more easily do cortical focal symptoms come on (from pressure or action from a distance).

Tumors of the corpus callosum have few characteristic signs. Bristowe gives the following: 1. Gradual increase of the symptoms. 2. Absence of, or slight, general symptoms of tumor (?). 3. Severe disturbance of the intelligence, stupidity, sopor, and a non-aphasic speech disturbance. 4. Hemiparetic symptoms, often combined with a less pronounced paresis of the other side of the body. 5. Absence of all cranial nerve symptoms.

Bruns and Giese have arrived at similar results.

Tumors of the *quadrigeminal region* are characterized by the following symptoms: paralysis of ocular muscles, particularly bilateral paralysis of the same ocular muscles, incoördination in walking and standing, impaired hearing, and probably amblyopia. The same symptoms occur in neoplasms of the pineal gland; these may also produce trochlear paralysis and nystagmus. Superior alternating hemiplegia is the focal symptom of tumors of the *cerebral peduncle,*—*i.e.,* crossed paralysis of the oculomotor and of the extremities. In tumors of this region, an intention tremor is often noticed, also a tremor of the type of paralysis agitans. A combination of oculomotor paralysis with hemiataxia of the other side is more rare (seat of the tumor particularly in the tegmentum, according to Krafft-Ebbing). In its further course, the third nerve of the other side may become involved. Collins has described a tumor of the aqueduct of Sylvius.

Tumors of the cerebellum are generally not hard to diagnose. The headache is most pronounced in the *occipital* region, also in the *back of the neck*, and even in the upper part of the cord. Frontal headache may exist conjointly. A slight degree of *nuchal* rigidity is often found. General and unilateral convulsions, and particularly attacks of opisthotonos and tetanic rigidity of the whole body also occur. Papillitis comes on early and is very prominent. *Vertigo* and *incoördination* are the most important, though not constant focal symptoms. In tumors of the vermis they are hardly ever absent. The patient complains continually of vertigo, or it occurs in attacks and particularly on change of position. Incoördination becomes noticeable on walking,—the patient reels easily and staggers from one side to another like a drunken person. It increases sometimes upon closing of the eye. The patient may fall from it, but this is by no means the rule. While this uncertainty in gait is a frequent symptom of cerebellar tumors, ataxia of the extremities occurs more rarely. It may be limited to one side of the body.

Vomiting is an almost regular symptom of cerebellar tumors, coming on early and remaining throughout the course of the disease.

We have a number of important symptoms which are due to tumors of the cerebellum pressing downward and forward upon the *pons and medulla oblongata*, and injuring the cranial nerves arising from these. If its seat is at the base of a cerebellar hemisphere and at the anterior part, pressure-symptoms on the part of the trigeminus, facial, acoustic, etc., may be the first symptoms noticed. These are persistent neuralgia of one side of the face, with anesthesia, trophic disorders, etc., paralysis of the facial (with disturbances of the electrical excitability) and of the acoustic, etc. Pneumogastric and laryngeal disorders may also occur, according to the seat of the tumor. It may be difficult to determine to what extent the symptoms are indicative of an involvement of the nerve-roots, and to what extent of an affection of the medulla oblongata. A unilateral involvement indicates more an involvement of the nerves. In the case illustrated by Fig. 238 I was able to establish an exact diagnosis from the symptoms of paralysis of the cranial nerves.

Symptoms of irritation—as, for instance, convulsive tic—may precede the paralysis. In a cerebellar tumor which pressed upon the medulla oblongata, there were present in the last months continued clonic twitchings of the velum palatinum, larynx, vocal cords, etc. Compression of the pons may produce a paralysis of the eyes towards the side on which the tumor lies; hemiplegia alternans may also occur in this way. Nystagmus is not a rare symptom of cerebellar tumor. Pressure upon the medulla oblongata may evoke respiratory disturbance. When this has once set in, death is not far off. The hydrocephalus accompanying cerebellar tumors may give rise to errors. Pronounced hydrocephalus may press the floor of the third ventricle forward so as to compress the chiasm. This can cause complete blindness. I have repeatedly observed *anosmia* as a symptom in cerebellar tumors (unilateral or bilateral); it is due to pressure upon and flattening of the olfactory nerve. The consciousness may remain intact a long time in cerebellar tumors. The knee-reflexes may be abolished in cases of cerebellar tumor. It may be a complication of cerebral disease with a spinal malady (tabes, sarcoma, gliosis, etc.). It has, however, also been noticed in uncomplicated cases of cerebellar tumor (more rarely in tumors of other regions). It cannot be definitely determined whether it is due to increased intracranial pressure or to toxic influences acting upon the posterior roots and tracts.

Tumors of the Pons and Medulla Oblongata.—Tubercle and glioma are particularly prone to establish themselves in this region. Tubercle may remain latent a long time. As a rule, however, pontile tumors induce marked symptoms, the focal ones being more pronounced than the general phenomena. Papillitis is comparatively often absent. The typical focal symptom is a *hemiplegia alternans;* the facial, abducens, and trigeminus, or one of these cranial nerves, upon the one side, and the extremities of the other side being paralyzed. Associated paralysis of the ocular muscles may also occur upon the side of the tumor. In other cases the above-mentioned cranial nerves are bilaterally affected. Bilateral hemiplegia may also occur; dysarthria and dysphagia are then almost regular symptoms. General unilateral or alternating convulsions are more rare. Sensory disorders and hemiataxia are observed with pontile tumors. In one case facial paralysis was the only symptom of a pontile tumor (Hunnius).

Tumors of the *medulla oblongata* may also run a latent course, particularly when they extend towards the fourth ventricle or develop within it, as is often the case with cysticerci. On the other hand, they produce symptoms corresponding in many respects to those of pontile tumors, except that they involve particularly the eighth to the twelfth cranial nerves. Deafness, deglutitory paralyses, dysarthria, aphonia, etc., are the chief symptoms. Other phenomena are cardiac and respiratory disturbances, singultus, and in many cases glycosuria, vasomotor symptoms, etc. The extremities may be paralyzed on one or both sides. Cerebellar ataxia is often noticed. The general symptoms of cranial pressure are often only slightly developed.

Tumors of the base of the brain are generally easily recognized. If they arise from the bones, they may break through into the nasal orifice or pharynx, and reveal themselves by hemorrhages in these regions or by becoming palpable; or bloody masses containing particles of the tumor may be discharged; or a sensitiveness to pressure of the part of the base of the brain accessible to palpation may be found. Choked disk is often absent, as is also vomiting. The cranial nerves are involved at their origin, or later in their course in varying number and grouping, being either compressed or infiltrated by the tumor and caused to atrophy. Tumors of the *hypophysis* cause bitemporal hemianopsia, unilateral or bilateral amblyopia, or amaurosis, or unilateral amaurosis with hemianopsia of the other eye. The background of the eye may remain normal for a long time; optic atrophy may develop later. Paralysis of the ocular muscles frequently occurs; exophthalmos and anosmia occasionally. Diabetes mellitus and insipidus also occur. The relations of tumors of the hypophysis to acromegaly will be discussed under the latter. In tumors of the middle cranial fossa the nerves leading to the orbits are particularly affected; trigeminal symptoms are very prominent.

In a case of this kind an exophthalmos with slight symptoms of paralysis of the oculomotor nerve, but with contraction of the pupil, was the first objective sign, though headache had preceded it for eight years. A sensory disturbance in the first trigeminal area came on, which was first recognized by absence of the corneal reflex, also amaurosis of the eye of the same side, and temporal hemianopsia of the other eye, and, finally, sensory and visual aphasia due to compression of the basal surface of the temporal lobe. The tumor was accurately localized. In another case the pressure which the tumor exerted upon the first trigeminal branch near the eyeball excited, by reflex action, twitchings in the left facial.

In tumors of the posterior fossa, etc., the cranial nerves may be affected on one or both sides. Pressure upon the medulla oblongata and pons can cause symptoms of compressive bulbar paralysis. These extradural tumors do not involve the sensorium, or involve it only in the terminal stage; they do not produce cerebral focal symptoms at all, or do so only in the last stage. It may be very difficult to decide whether the tumor arises from the lower surface of the cerebellum and presses forward towards the base of the brain, or whether it arises from the bones or membranes and grows into the cranial space.

An important aid in topical diagnosis is the *sensitiveness of the cranium upon percussion.* In some cases slight percussion on every part of the head will be painful. I found this phenomenon whenever the cerebral pressure was pronounced and the cranial bones thinned by osteoporosis. It is not dependent upon the location of the tumor. In other cases percussion does not reveal sensitiveness on any part of the head. There remains a relatively small number of cases in which percussion produces pain at some circumscribed part of the cranium. This indicates most probably a relationship between the tumor and this part of the cranium. Peripheral tumors are particularly apt to cause this symptom.

The temperature of the skin upon that part of the cranium which lies over the tumor is sometimes increased.

Percussion over the cranial region where the tumor lies may produce a tympanitic or cracked-pot sound (Bruns). It may occur under other conditions also, and is always present in sucklings. (See the chapter on Aneurisms for the value of the so-called cerebral murmurs.)

Diagnostic Points.—Cerebral tumors may remain latent until death. This is particularly true of slowly progressive or entirely stabile tumors as psammoma, lipoma, cholesteatoma, and cystic tumors. Every persistent and severe headache should awaken a suspicion of brain tumor. Such headaches are often due to other causes, as is well known. A papillitis or optic neuritis almost confirms a diagnosis of cerebral tumor. Endeavor to exclude meningitis, cerebral abscess, hydrocephalus, and nephritis, as these produce a form of retinitis very similar to choked disk. If pronounced chlorosis is present, the detection of papillitis does not suffice to refer a severe headache to tumor. With chronic lead intoxication present, it is also not a certain sign of cerebral tumor. Optic neuritis also occurs in multiple neuritis, and probably in the alcoholic as well as in the cachetic types. When this is present in cases of multiple neuritis, or when the optic neuritis is due to alcoholism, the visual disorder is generally a characteristic one (central scotoma for colors).

If papillitis is not present, if the opthalmoscope reveals nothing, the diagnosis of cerebral tumor is not yet shattered. It may be permanently absent at the beginning and also in small, cystic, and superficially lying, extracerebral tumors, and be missing for a long time in those arising in the motor zone, pons, and medulla oblongata. When the result is negative, endeavor to acquaint yourself more exactly with the nature of the headache. Seek after other causes, after toxic factors (arsenic, lead, copper, mercury, morphine, nicotine), arteriosclerosis, circulatory and digestive disorders; obstipation is often a cause. It is particularly important to determine whether hemicrania, hysteria, or neurasthenia is present. Although hemicranial headache is characterized by its periodic occurrence, in some cases it becomes permanent at a later stage. An exact anamnesis will establish the diagnosis. Hysteric, more often, however, neurasthenic, headache may furnish diagnostic difficulties. The discovery of hysteria or neurasthenia means much, but not all. But the determination of the fact that the headache is dependent directly upon psychic excitement, increasing on emotion, disappearing when the attention is distracted, helps to make the case clear.

If the headache indicates a neoplasm of the brain, while the ophthalmoscopic examination is negative, the diagnosis may be based upon other criteria. Stupor and sleepiness are important signs, but there are other conditions—above all uremia and other intoxications, as well as the psychoses which cause sleep and dream-like conditions—which must be excluded. Vomiting is only of importance for diagnosis purposes when it is of a cerebral character. Retardation of the pulse is of more meaning; only arteriosclerosis should be excluded, and it should not be forgotten that the pulse may be markedly retarded at the height of an attack of migraine. This also occurs in melancholia. Vertigo is a very vague symptom. Its nature should, therefore, be exactly determined upon. Vertigo in cerebral tumor occurs independently of conceptions, phobias, etc., comes on without any motive, and is permanent. The different forms and causes of vertigo are discussed in full at another place. The *focal symptoms* are of much value when they develop gradually and progress constantly or *gradatim*. This is, however, no axiom. A tumor may remain latent a long time, or only cause general symptoms until a hemorrhage into it or a circumferential softening produces paralytic and irritative symptoms of an acute, apoplectic character. This is, however, so rare that focal symptoms may be regarded as the rule. As abscess may take a similar course, it must be excluded. Meningo-encephalitis (syphilitica and tuberculosa), as also that rare disease, chronic cerebral softening, may cause similar focal symptoms. Great caution is necessary in the consideration of cortical epilepsy as a symptom, as it also occurs in hysteria, intoxications (particularly alcoholism and uremia), paralytic dementia, etc.

If the diagnosis of cerebral tumor has been definitely established, its character must be looked into with the help of the points given above and the following questions: Is there tuberculosis of another organ present, or is there a pronounced predisposition to it? Is the patient infected with syphilis? If the anamnesis and physical examination are negative, syphilis is by no means excluded. The clinical picture and treatment may reveal much. If cancer or sarcoma is present in other organs, it is probable that the brain tumor is of that character. Do not neglect to examine the lungs, as a somnolent patient, neither by answers to questions nor from complaints, directs attention to this organ. It is also advisable to examine the nasal, pharyngeal, and laryngeal structures.

Another question to decide is the localization of the tumor in the brain. Of only a scientific interest in former years, a local diagnosis in the last decade has become of not inconsiderable practical importance. Still, to-day, however, we are only able in a small percentage of cases to localize a tumor exactly. We first seek for focal symptoms, which are of more importance the earlier they set in. If we are called to see the patient when he is in the somnolent stage, with all symptoms of intracranial pressure, the focal symptoms if present have a localizing value when they have been present from the beginning. A tumor which produces focal symptoms of the motor zone can be most easily localized. We have no trustworthy criteria, however, enabling us to distinguish cortical neoplasms of the motor zone from those of the subcortical medullary layer. The cranium should be carefully percussed, as a place may be found which is sensitive to slight percussion.

Confusion with paralytic dementia is only possible in a few cases. These are the ones which commence with persistent headache and with paralytic and irritative symptoms whose "paralytic" nature cannot be recognized without other factors. Papillitis does not, however, occur in dementia paralytica, and not simple stupor but true dementia is found present. The attacks, also, even when they resemble those of cortical epilepsy, do not leave behind any continued and complete paralyses, but, on the contrary, generally increase the disturbance of intelligence, etc. The pathognomonic speech disturbances of paralytics and the other symptoms of this disease are also aids in making a diagnosis.

Cerebral tumor not rarely produces a clinical picture similar to that of multiple sclerosis (see page 214). Confusion is particularly possible when either a slight neuritis is present or the choked disk has already passed on to atrophy. If nystagmus also occurs, which is very probable in cerebellar and quadrigeminal tumors, and an indefinite tremor, as is observed at times in cases of cerebral tumor, and in hydrocephalus, and also headache, vertigo, and an uncertain gait, sclerosis cannot help but be thought of. This error can, however, I think, be avoided by a careful examination. The visual disorder is, as a rule, more marked in the atrophied eye than is the case with sclerosis; the tremor may resemble that of multiple sclerosis, but is generally inconstant and more indefinite, consists of finer, more rapid vibrations, and is not so markedly connected with voluntary movement. The tremor in cerebellar and pontine tumors may resemble most an intention tremor, but is in such cases usually combined with ataxia. In one of my cases in which upon movement the tremor resembled that of sclerosis, a tremor in the other muscular groups was noticed when the patient was at rest. Stupor does not belong to the symptoms of sclerosis; it may, however, be absent for a long time in cases of tumor, particularly in cerebellar ones; spasms and retardation of the pulse speak against sclerosis, and also vomiting (I have seen it appear temporarily in a few cases of vertigenous attacks of multiple sclerosis), aphasia, cortical epilepsy, etc. Finally, spinal symptoms are rarely absent in disseminated sclerosis.

Acute encephalitis, which occasionally occurs with optic neuritis, differs from tumor in its acute, febrile development and in its further course. In some cases, though, a differential diagnosis cannot be made at the beginning. (Consult the chapters on hydrocephalus and abscess for a differential diagnosis from these ailments.)

Lumbar puncture (page 478) may be of value when it is desired to differentiate cerebral tumor from other diseases which do not increase the quantity of cerebrospinal liquid. If increase in quantity and pressure occurs, a microscopical, chemical, and bacteriological examination may enable us to discover evidence of a purulent or tubercular meningitis. If a clear liquor, free from leucocytes and bacteria, is found, these conditions cannot be entirely excluded, but, on the whole, all that is necessary is to determine whether hydrocephalus or tumor is present. This question is not positively decided by the liquor, but the quantity of albumin is generally larger in tumor than in cases of chronic primary hydrocephalus.

It should also be remembered that the result of spinal puncture may be negative in cerebral tumor, particularly when the ventricles, and the cranial and spinal cavities are shut off from each other. A lumbar puncture may often then increase the cerebral symptoms.

My attempts to use the X-rays for diagnostic purposes in these cases failed. I was able to experimentally determine that a tumor placed within the cranium upon the brain is very distinctly noticed, but attempts on living subjects did not secure positive results. It is well-known that shot within the cranium and brain may be detected in this way (Eulenburg, Bergmann, Scherning).

Etiology.—The causes of cerebral tumor are not altogether known. We know that a *trauma* affecting the cranium may induce it. The initial symptoms may come on soon after the injury but often are only observable after some time. Some forms of tumor are due to congenital developmental anomalies; this may also be true of glioma. Cicatrices may probably form the starting-point for tumors. The *infectious* nature of some and the metastatic nature of other tumors indicate their etiology. That actinomycosis has also been found in the brain has been already mentioned.

Course and Prognosis.—The course is almost always a chronic one. It lasts, as a rule, for from two to four years, though it is not uncommon for it to extend over a period of from five to six or even ten years. I observed such an insidious course particularly in tumors of the cerebellum, right frontal lobe, and base of the brain. Experience has shown that death does not occur so soon in young persons with elastic cranial walls and sutures as in adults. The course in solitary tubercle, glioma, and osteosarcoma is slower than in soft sarcoma, and particularly than in carcinoma; it is especially protracted in some cases of cholesteatoma and cavernous angioma.

The tumor often remains latent a long time, and then under acute and even stormy symptoms rapidly causes death.

The *prognosis* is comparatively good in *syphilitic* cases; grave in other forms. *Spontaneous recovery* is possible with echinococcus, cysticercus, and aneurisms. Some authors believe that tubercle is capable of spontaneous metamorphosis and retrogression. A periodic improvement is occasionally produced by a part of the ventricular contents, after wearing away of the cranium, being evacuated externally, especially through the nose.

Treatment.—If syphilis is present, or the symptomatology indicates a syphilitic neoplasm, the method of treatment is already laid out for us. It cannot be too strongly insisted upon, however, that the slightest suspicion in this direction demands an *antisyphilitic* treatment. If the clinical picture has no similarity with that of cerebral syphilis, and if the anamnesis and physical examination in addition offers no indication of syphilis, no time should be spent in an antisyphilitic treatment, providing that an operation at the proper time can be expected to relieve the patient. This holds true, however, in only a few cases. On the other hand, continued administration of potassium iodide in large doses should not be omitted in any case, as the use of this drug, even in antisyphilitic tumors, has been known in some cases to result in pronounced improvement and in intermissions.

Operative treatment of cerebral tumors reveals to us one of the most remarkable advancements in therapeutics. It was not many years ago that every person suffering from cerebral tumor was thought to be doomed to an early death. Wernicke, in 1881, advanced the indications for operation, and soon after published a case in which a suppurating tubercle of the occipital lobe was found upon opening the skull and evacuated. American and English surgeons, however (Bennet, Godlee, Macewen, and others), showed that it was possible to extirpate a cerebral tumor without danger to life, and Horsley, more than all others, proved that operative removal of tumors, if the seat has been definitely and early localized, can result in complete recovery.

Further experience showed that only a *small* percentage of cases (six in one hundred) were accessible for this treatment, and also that the danger to life is not a slight one.

The indications, as given by Bergmann and, after him, by others, are about as follows:

The tumor must be located in the cortex or near it, on the outer convex surface of the hemispheres; it must not be diffusely lost in the brain-substance, but should be enucleable. Diffuse neoplasms have, however, been extirpated with success. The probability must be that only *one* tumor is present, and that this one is not of a malignant or of a metastatic character. It should not be too large. Finally, one must be able to locate it from the symptoms.

Practically, the matter stands thus: tumors of the motor zone are about the only ones that can be early localized and diagnosed. Most of those that have been operated upon, and particularly those that were successfully operated upon, were of this region. Even though we are unable to definitely determine whether the cortex or the subcortical area is the seat of the tumor, we know that it is a relatively superficial, and therefore an accessible, region. The earlier the diagnosis is made the more probable is it that the tumor is a small one, and therefore the better are the chances. Erb's case, however, shows that large tumors of this region may also be enucleated with success, or, if the extirpation was a partial one, that the operation can be repeated several times. Bramann successfully removed a tumor weighing two hundred and eighty grams from the frontal brain. In a case operated upon by myself and Köhler,—probably the first treated in this way in Germany,—although we succeeded in removing only a part of the tumor, we secured pronounced improvement for a period of six months. The patient was delivered of a living child in the course of this half-year.

Many of the focal phenomena may furnish a basis for operation. If, in addition to the general symptoms of tumor, we find signs of a pure, well-pronounced form of aphasia, if we discover that this symptom was present from the beginning, even though only slightly, and has gradually progressed, we are justified in opening the cranium in the region of the third left frontal or first temporal convolution and removing the tumor. The discovery of sensitiveness of a part of the skull on percussion may aid materially in localizing the tumor. Hemianopsia can only rarely be referred to the cortex with sufficient exactness to justify operative treatment. Its combination with alexia or with visual aphasia indicates the possible seat of the tumor, but further observation is necessary to regard it as an indication for operation.

Surgical treatment of cerebellar tumors offers such slight cha a cure that an operation is rarely justifiable. (Macewen and Parkin have each had a successful operation.)

If a cerebral tumor is inoperable, which is true of the majority of cases, the question arises whether we have no medicaments which can act symptomatically and relieve the chief symptoms, particularly the headache. *Morphine* is our chief aid, and in severe hopeless cases there should be no hesitation in using it. I have seen *antipyrin* lessen the headache in several cases. The other narcotics may also be used, alternating with morphine. An ice-bag upon the head sometimes relieves the pain. Bloodletting or leeches may also be used when the pains are very severe. Hot foot-baths, purgation, etc., may also relieve somewhat.

Even in these incurable cases, however, surgical treatment may do good. A simple *trephining* of the cranium with opening of the dura and removal of some of the cerebrospinal liquor may lessen the pressure within the skull. This is sufficient to relieve the *headache*, *choked disk*, stupor, and other pressure symptoms for a long time. I have observed this result in several cases. In one case, in which the tumor could not be entirely removed, and later grew to a considerable size, a choked disk was absent until death. In such cases, however, an operation should only be undertaken when the symptoms are very severe, and cannot be ameliorated by any other method, or when the ocular disturbance progresses rapidly and blindness is threatened. Select a place on the cranium under which the tumor lies, or, in doubtful cases, the temporal or lower parietal region of the right hemisphere. The enthusiasm for operative treatment of cerebral cases has decreased considerably. The cases which can be cured are few, and these are exactly those in which the subjective symptoms are so slight that it is difficult to induce the patient to consent to an operation. The tumor has often not been found. On the other hand, the dangers of the operation are not inconsiderable. as Bergmann has lately shown in a convincing manner. The relatives should always be informed what chances the operation furnishes.

Tapping of the ventricles has been recommended for the relief of the symptoms, though no particularly good results have been secured. Beck has recently advocated it again.

Lumbar puncture may produce temporary improvement in some cases. I have noticed a longer amelioration in only one case. Heubner, Fraenkel, Goldscheider, and others have had here and there a good result. On the whole, however, the results were not very promising. On the contrary, in many cases some of the symptoms, especially the headache, increased after the puncture. I saw new paralytic symptoms almost immediately follow the operation in a case of quadrigeminal tumor, in which but little fluid was secured from the spinal canal. The tumor probably had closed the aqueduct of Sylvius. Spinal and girdle-pains have also been caused by the operation. Finally, it may cause death, as the observations of Fürbringer, Lichtheim, Wilms, Stadelmann, and others show. When done, it must be with great caution, under exact control of the pressure, with but slight evacuation of the cerebrospinal fluid at one time, and must not be repeated in cases where the communication does not appear to be open.

ANEURISMS OF THE CEREBRAL ARTERIES.

Excluding miliary aneurisms of the intracerebral arteries, the ordinary forms of aneurisms, while they are comparatively infrequent, occur much more often in the cerebral blood-vessels than in the arteries of the rest of the body. In the inside of the brain true aneurisms are rare. They manifest themselves more often in the arteries at the base of the brain before their entrance, or that of their branches, into the cerebral substance. The *middle cerebral* and *basilar* arteries are particularly often attacked. The bifurcations are favorite seats. Cerebral aneurisms are either fusiform or saccular, and may be either true or false, as those of other regions. Aneurismal dilatation occurs especially on the vertebral and basilar. The arterial tumor may vary in size from that of a pea to that of a hen's egg. The arteries on the left side are more often involved than those on the right.

The aneurismal formation is due to disease of the arterial walls, which causes a degeneration of its contractile elements at circumscribed spots or in larger areas; the arterial walls become dilatable and inelastic from this. *Atheroma* and, particularly, *specific arteritis*, alter the vessel in this way. *Emboli* of the cerebral arteries are extremely prone to produce aneurismal formation, as the embolus produces a partial closure of the vessel on the one hand and a local inflammation on the other. The importance of *cardiac disease* and *lues* is made more clear by the fact that aneurisms tend to occur in youthful and middle-aged persons, and are rarer in aged persons. *Traumata* (of the cranium) may also be an exciting cause.

Aneurisms often do not present any symptoms until the moment of *rupture*, which occurs in very many cases and produces death. It causes symptoms of cerebral hemorrhage, either slowly developing (ingravescent), or it takes a stormy course, the patient falling suddenly unconscious, with deep coma, general convulsions, paralysis, fever, asphyxia, etc.

The rupture need not end in death; it may repeat itself and entail paroxysmal attacks with severe symptoms (Kretz). Generally, however, either *general* or *local* symptoms of a cerebral disease come on. To the first belong *headache*, sometimes described as a pulsating one, *vertigo*, *vomiting*, *stupor*, and *convulsions*, which last, however, do not often occur. These symptoms are held in common by all diseases confined to the cranial fossæ, and which increase the pressure.

A *pulsating vascular murmur* is particularly characteristic of aneurism, and may sometimes be heard from a distance. It may also be due to tumors which arise in the vicinity of a large artery and compress it (Hennig, Jurasz, etc.); also to very vascular tumors. In childhood these murmurs are of no pathognomonic importance, as they occur in healthy children, in rachitis, anemia, hydrocephalus, etc. In adults, also, they may be caused by anemia and by vasomotor disturbances. A congenital narrowness of the carotid foramen may also be a cause (Troeltsch, Urbantschitsch). Cephalic murmurs have been detected several times in exophthalmic goitre; also in compression of the sympathetic nerve by tumors.

Compression of the common carotid generally stops a vascular murmur due to aneurism of the external carotid or its branches.

The local symptoms are dependent upon the seat of the aneurism, and, as a rule, point to a process at the base of the brain.

Aneurisms of the *internal carotid* may compress the optic nerves, the nerves of the eyeball, the olfactory, and the first trigeminal branch. They cause visual disorders with or without an ophthalmoscopically observable lesion, exophthalmos, pain, hyperesthesia, and hypesthesia of the first trigeminal region, oculomotor paralysis, and anosmia. Pressure upon the cavernous sinus may produce a hyperemia of the retinal veins (?) and a dilatation of the facial veins. If the tumor is large and on the left side, the speech centre may become involved, and the motor paths may be impaired by action from a distance, or the cerebral peduncle may be directly compressed, thereby causing hemiparetic symptoms. The blood, if rupture occurs, may reach the cavernous sinus and produce a pulsating exophthalmos. Aneurisms of the middle cerebral, basilar, posterior communicating, and the other cerebral arteries produce symptoms referable to the part involved.

Aneurisms of the corpus callosum artery endanger particularly the optic and olfactory. In one case a unilateral temporal hemianopsia was said to have been one of the symptoms.

Aneurism of the artery of the Sylvian fossa often remains latent until death ensues. It may injure the oculomotor and olfactory, but acts particularly upon the brain-substance and by compression produces aphasia, monoplegia, hemiplegia, etc. Motor symptoms of irritation generally precede the paralysis.

Aneurisms of the posterior communicating artery may cause hemianopsia by compression of the optic tract.

Aneurisms of the *basilar artery* press against the pons or against the cerebral peduncle or the medulla oblongata. The pons may be considerably excavated, the cranial nerves running here may be compressed and torn and even atrophied. The patient complains of headache in the back of the head, which is often made worse by movements of the head. Compression of the pons, etc., evokes the phenomena of *hemiplegia alternans* or of *bulbar paralysis*, which develop by easy stages or in a subacute manner. We find, in addition, marked symptoms of irritation and paralysis in regions innervated by other nerves (fifth, seventh, tenth, twelfth, etc.).

Aneurisms of the vertebral produce a similar symptom-complex. (See the chapter on Acute and Compression Bulbar Paralysis.) Several times in aneurisms of the posterior cranial fossa it was noticed that forward inclination of the head evoked severe respiratory disturbances, particularly cessation of breathing. To a certain extent, a step-like course is noticeable in this form also.

The prognosis is very grave. In most cases death occurs after some months or years. Spontaneous recovery is occasionally noticed.

Treatment.—Potassium iodide and mercury should be given if syphilis is expected. Continued use of potassium iodide may do good in any case, no matter what the cause is. Ergot is not of much benefit. Everything which increases intracranial pressure or promotes venous congestion should be avoided. A light, unirritating diet should be prescribed. Alcohol, coffee, and tea must be eschewed. Laxatives are advisable. The head must not be laid low, or bent too far forward or backward. Aneurisms of the *internal* carotid have several times been successfully treated by ligating the common carotid.

Lumbar puncture is never permissible. In one case it produced aneurismal rupture and death.

THE PARASITES OF THE BRAIN.

Cysticercus cellulosæ is a parasite that is often found in the brain; echinococci occur more rarely. The cysticerci are generally very numerous, occurring in the arachnoidal and pial meshes, in the cerebral fissures, rather often in the ventricles, swimming free or attached to the ependyma, and rarely in the deeper structures. They vary from the size of a pea to that of a walnut. The formation of daughter vesicles causes a branched, grape-like formation, *cysticercus racemosus*, which may reach a larger size. On closer inspection the head and neck of the worm is seen as a small, dark point, which on microscopic examination reveals a hooklet and four suckers. If the cysticercus dies, the contents of the vesicles may calcify. A single cysticercus is rarely observed.

Echinococci occur alone or in numbers, even a hundred vesicles have been found. They have a superficial seat on the apex as well as at the base of the brain, in the medullary substance, and at times in the ventricles. Their size varies from that of a pea to that of a man's fist.

Symptomatology.—It is not uncommon to find *cysticerci* post mortem, though no symptoms had indicated their presence. Even when they occur in the fourth ventricle, they may remain latent to within a short time of death. Sudden death without any premonitory signs occurred also in cases where they had their seat in the third ventricle.

As a rule, however, they cause symptoms of a variable character, and which often do not even denote a cerebral disease. A headache of a paroxysmal character, a transitory vertigo, loss of consciousness for a short time, with some vague subjective troubles, cause a diagnosis of hysteria, neurasthenia, hemicrania, vertigo, epilepsy, etc., to be made, until finally severer cerebral symptoms come on, and a grave disease is suspected. In many cases these severe symptoms come on from the beginning; but even then satisfactory criteria upon which a diagnosis of cysticercus can be based are lacking.

Convulsions are a prominent symptom. They may resemble those of epilepsy; more often the attacks are like cortical epilepsy, and the picture is still more often a variable and *mixed* one. Now it is simple unconsciousness, now a condition of confusion and distraction. Twitchings are generally present at the same time, which play in one extremity or in one *muscular group, vary their location,* shift from one side to the other, etc. These twitchings may last hours and days, or be very fleeting. Twitchings in the region of the sternocleidomastoid, trapezius, omohyoid, facial muscles, etc., are particularly often noticed. These attacks are so indefinite and the loss of consciousness is so slight that they are very similar to hysteric attacks, especially as a general tremor, a deglutitory spasm, or a singultus may precede or follow them. According to Wernicke, epilepsy caused by cysticerci is particularly liable to pass into a lethal state.

Psychic disturbances are often observed; not a few of the observations published have come from insane asylums. Simple imbecility is generally seen; at times we find *conditions of excitation,* hallucinatory delirium, a persistent stage of *confusion* which, when it appears in combination with spasms, is thought to represent a psychic equivalent, or it may, in combination with the imbecility, simulate the picture of paralytic dementia. *Focal symptoms* are not rarely present—hemiparesis or hemiplegia, monoplegia, aphasia, etc. These also are only temporary. If a cysticercus is seated in the fourth ventricle, *glycosuria, respiratory and circulatory disorders, cerebellar ataxia, vomiting,* and various paralyses from compression of neighboring parts are evoked. If the cerebellum—as happens in not a few cases—is involved, occipital pain, vertigo, and reeling are the most pronounced symptoms. Headache and vertigo are the most constant symptoms of all forms. The cranial nerves may become involved and produce visual disorders, deafness, etc. A pronounced choked disk is not often seen. If the cysticercus has its seat, for example, in the fourth ventricle or in the aqueduct of Sylvius, the hydrocephalus produced thereby may cause a simple amaurosis without any pathologic lesion. The symptoms generally point to a disease-process in several parts of the brain, which causes symptoms of irritation and paralysis, mostly the former, which is not steadily progressive but undergoes remissions, so that periods of complete good health may intervene between the attacks. The clinical picture, therefore, resembles that of the neuroses and psychoses on one hand, that of cerebral tumor on the other hand. The irregularity and variability of the symptoms are supposed to be due to the power of locomotion possessed by the cysticercus. Griesinger says that a cerebral disease with a paralysis present from the first, or coming on early, is not due to cysticercus. Persistent and progressive paralysis especially is rare in this disease.

The above data enable us now and then to establish a probable diagnosis. The presence of cysticerci in the skin supports it if, upon search, they are found. These are tumors of the size of a *pea* or a *hazel-nut* (rarely larger or smaller), which are present *under the skin or in the muscles*, are *movable*, and feel *elastic* and as hard as *cartilage*. Excision and examination will confirm the diagnosis.

The cysticerci are occasionally found in the *eye*, and may be seen with the ophthalmoscope.

In addition, another help in making a diagnosis is the determination of whether an opportunity for infection had occurred, whether a tapeworm (tenia solium) is present, and whether the occupation or manner of living (eating of raw ham) has made an invasion of cysticerci possible. It can hardly be possible to refer symptoms caused by intestinal parasites to cysticerci cerebri, although Peiper has lately established the hypothesis that the animal parasites produce toxins in the human body, capable of causing the most varied cerebral phenomena. I have repeatedly been able by the detection of cysticerci in the skin, as have also others, to diagnose correctly cerebral cysticerci; while in those cases in which the cysticerci of the skin were absent cysticercus cerebri could not be diagnosed. One of my patients, for instance, complained of headache and uncertain gait. She acted like a hysteric person; reeled when walking, but with a cane walked well, without supporting herself upon it, or walked without any difficulty when it was suggested to her that she could walk. At first a slight papillitis was thought to have been seen; later on it could not be detected. The absence of all objective symptoms and the peculiar psychic disposition of the patient made me diagnose hysteria as the most probable disease present. One day the patient became unconscious and died, and the post-mortem revealed a large cysticercus in the roof of the fourth ventricle. In another case, a cysticercus of the fourth ventricle produced a paraplegia of all four extremities; the brachial paralysis disappeared later on. Mental confusion and incontinence of both urine and feces came on later. If the anamnesis and psychic condition had been ignored, a myelitis would have been diagnosed. Cysticerci may also develop simultaneously in the brain and in the spinal cord.

The prognosis is grave, though a standstill and recovery are not impossible, as calcified cysticerci have repeatedly been found in the brain. I treated a man for many years for cortical epilepsy, which, on account of the cysticerci of the skin present was supposed to be due to cerebral cysticerci. The attacks finally disappeared, and the patient was discharged as cured.

The treatment is in the first place prophylactic. Raw ham must be avoided. A tenia must be removed and made innocuous as soon as possible. A symptomatic treatment is all else that can be done. It may be thought that a cysticercus of the motor zone which causes symptoms of brain tumor could be extirpated with good results; generally, however, the multiplicity of the cysticerci renders this hope futile. Just after this chapter had been written, the Breslau surgical clinic reported a case, in which a cysticercus had been found in the motor zone and extirpated. Certain symptoms, however, persisted from which the multiplicity of the cysticercus could be decided upon.

Echinococci cerebri produce, as a rule, symptoms of cerebral tumor, of which in a case of mine only the choked disk was absent. In these cases, movements generally increase the headache, and the patient has often the feeling as if something were moving in his head. Westphal's case is interesting. In it, the tumor *broke* externally through the cranial bones and towards the nasal cavity, wore away the bones, and formed palpable spaces, out of which the fluctuating tumor protruded. Probing made the diagnosis certain. Ninety echinococcus sacs emptied themselves in this manner and recovery thereupon occurred. Another similar observation is known in literature.

Recently observations have increased in which the echinococci have been found to produce symptoms of a tumor of the motor zone and made an operation seem advisable (Hammond, Fitzgerald, and others).

An embolus due to echinococci of the cerebral arteries has also been observed; the echinococci probably came from the left ventricle.

HYDROCEPHALUS.

Hydrocephalus is mostly a *congenital* disease. It may, however, be *acquired* at any age. It seems advisable to us to describe separately the congenital and the acquired form.

The cause of *congenital hydrocephalus* is not yet clear. Traumata affecting the pregnant woman and psychical excitement have been given as causes, though their influence is entirely hypothetical. Cachexia, alcoholism, and particularly syphilis have been blamed with more right. Bärensprung, Sandoz, Heller, and Heusinger have made observations of this kind. I observed a moderate hydrocephalus several times in hereditary syphilitics, severe symptoms only coming on later in life. It is also certain that a family predisposition is responsible to some extent, several children in the same family being afflicted with hydrocephalus, and their descendants again being born with the same disease.

Congenital hydrocephalus consists in the collection of excessive quantities of water in the cerebral ventricles. The pathogenesis is not exactly known. It is assumed that an *inflammation of the ventricular ependyma* is the cause of the transudate, others believe that it is due to a *displacement* of the *communicatory orifices* which connect the ventricles with each other and with the subarachnoid space. That this may cause hydrocephalus is evident; we do not know, however, whether and how often these moments are in question. Luschka, Monro, Neurath, and others have described such a pathologic condition. Huguenin desires to distinguish an inflammatory and a dilatatory hydrocephalus. We speak of an *internal* and an *external* hydrocephalus, according as the fluid fills the ventricle or the subarachnoid space. The latter is not so important as the former and is generally a result of some other cerebral disease (*hydrops e vacuo* after atrophy, etc.), and will not be fully considered here.

The fluid, which is generally clear and colorless, and which contains little albumin and a slight quantity of salts (NaCl, etc.), dilates all, but especially the lateral ventricles. If the avenues of communication between the ventricles are displaced, the hydrocephalus may confine itself to certain ventricles. The fourth ventricle is the least often affected. The quantity of this fluid varies from a few ounces to many litres; the average is about one litre.

The brain-substance is almost regularly injured. It is thinned out more or less, even to such a degree as to reduce the wall of the hemisphere to a few millimetres in thickness. The convolutions and sulci may be completely obliterated, while the cerebral mantle which holds the fluid resembles a thin, flapping sac. The central ganglia are often flattened and the floor of the third ventricle puffed out. The ventricular ependyma appears granulated, and inflammatory alterations of the choroid plexus are occasionally noticeable.

The *cranial circumference* is always increased. It is normally from about thirty-five to forty centimetres in the new-born, and reaches forty-five centimetres in the first year. In hydrocephalus it may be sixty, eighty, or even one hundred centimetres, and in one case is said to have been one hundred and sixty-seven centimetres. The cranium becomes *rounded* and the *frontal* and *parietal prominences* stand out prominently. The orbital roof is depressed downward (Fig. 240). The cranial bones are mostly thinned, even to the thinness of paper; the sutures are separated; the fontanelles remain open a long time; they have been found open in the third decade in some cases.

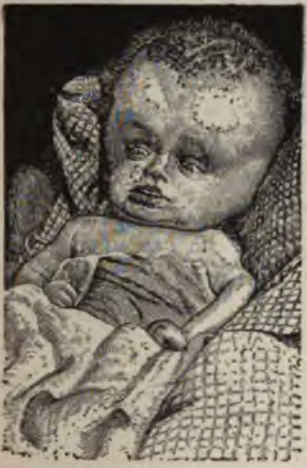
Symptomatology and Course.—If hydrocephalus is completely developed at birth, it may hinder delivery, and many individuals affected in this way die intra partum. Generally, however, it only develops in extra-uterine life. The cranium, not much enlarged at birth, gradually grows larger and larger. The increase may amount to one centimetre a week or more. The characteristic signs of this disease are alterations of the cranial circumference and the cranial form on the one hand and anomalies in cerebral functions on the other. The disproportion between the skull and face is marked. The enlargement of the first at times involves particularly the sagittal diameter; the skull becomes pronouncedly dolichocephalic. The eyes are pressed downward. The veins appear prominently. The hair on the head grows sparsely. The thinning of the cranial bones is sometimes recognized by palpation. They may be so transparent that the vessels may be seen shimmering through them. Auscultation reveals sometimes a loud vascular murmur. The fontanelles are wide and prominent, and the sutures gape.

The most important and constant cerebral symptom is the defective intelligence. Most of the hydrocephalic children are *idiots* or *imbeciles;* they do not learn to speak, or only late in life, and then only partially. In some cases, however, the intellect is not weakened very much, and in a few cases is normal (Fig. 241).

The *motor functions* are more or less impaired. The children learn to walk late and with difficulty or not at all. Complete hemiplegia is rare. The trunk is shrunken, the arm movements are awkward, uncertain, and weak, the head cannot be held erect (partly on account of its weight), and *spastic symptoms* are often noticed in the legs. The incontinence of urine and fæces, which is often present, is probably due to the mental condition. In many cases *choked disk* or *optic atrophy* is present, though these symptoms are more often met with in the acquired form. Paralysis of the other cranial nerves and sensory disorders are rarely observed in congenital hydrocephalus. General spasms of an epileptiform character are not infrequently met with. Fever, stupor, vomiting, etc., occasionally occur in the course of the disease.

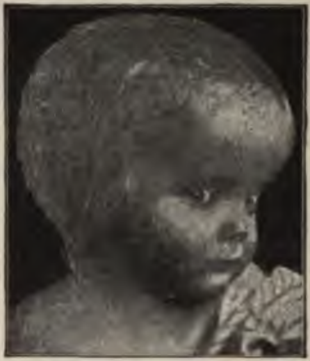
Developmental anomalies, as spina bifida, hare-lip, encephalocele, club-foot, dwarfism, etc., often combine themselves with hydrocephalus. I have seen it occur with albinism. Hydrocephalic children die, as a rule, soon after birth. The exceptions, however, are not rare, and one patient is said to have become seventy years old. Spontaneous evacuation has occurred several times, the fluid having passed through fissures in the cranial bones into the nasal cavities (more rarely into the orbital cavity), and improvement, even recovery, having followed. In a few cases a spontaneous perforation of the cerebral membranes, with effusion of the liquid through the sutures, has been observed. Huguenin has gathered nine cases of perforation, five of which recovered. Injuries of the cranial bones have also resulted in evacuation of the cerebral fluid.

FIG. 240.



Condition of the cranium, eyes, etc., in hydrocephalus. (After Hirt.)

FIG. 241.



Case of hydrocephalus with intact intelligence.

Differential Diagnosis.—The hydrocephalic head must not be confused with the rachitic skull. The latter is more four-cornered, box-shaped, the fontanelles are not prominent, rachitic symptoms are present, and cerebral phenomenon are lacking. Rachitis may be an exciting cause of hydrocephalus. Some normal individuals also have very thick cranial bones.

We find also certain peculiar congenital, or intra partum developed, *cranial malformations*, which may be combined with a neuritic or optic atrophy, without the cranial circumference being enlarged *in toto*.

There is especially a form in which the basal cranial area, particularly the anterior, is enlarged, so that the malar bones appear prominently, while the other circumferences of the cranium are perhaps less than normal (Tower skull). In such cases I observed that the facial veins were particularly prominent. The intelligence was normal. Whether in such cases hydrocephalus was in play, which, on account of other developmental anomalies of the bony cranium, did not appear prominently, I cannot say, though I did not observe increased pressure and increase of the cerebrospinal fluid in lumbar puncture. The optic trouble is said to be a result of hyperostosis of the basal cranial bones and the contraction of the optic foramen produced thereby. In one of my cases the deformity was, without doubt, the result of a birth trauma, due to a contracted pelvis.

The prognosis of congenital hydrocephalus is very earnest; only in mild cases can there be any possibility of life remaining and of grave disturbances not developing. Pronounced hydrocephalus greatly impairs the cerebral functions and almost always causes early death. Spontaneous breaking through externally is so rare that it need hardly be considered. The results of treatment are so unimportant and uncertain that from this aspect also the prognosis must be regarded as unfavorable.

Treatment.—All therapeutic endeavors formerly were directed to the removal of as much water as possible from the organism so as to promote an absorption of the fluid in the ventricles. *Diuretics* and *purgatives* were, therefore, freely administered, but such measures were futile and useless.

Some success has attended the use of potassium iodide and mercury (calomel inwardly, sublimate baths or inunction cures), even in cases where syphilis had not been present.

Another method of treatment is the prevention of further enlargement by *compression of the cranium*. Positive results have not been secured by this method, and such forced attempts must in themselves be regarded as dangerous. Trousseau recommended for this purpose strips of adhesive plaster attached from each mastoid process to the outer part of the opposite orbit, from the root of the nose to the external occipital protuberance along the sagittal suture, then in circles around the head, etc. Others favor a broad elastic band.

Surgical interference has not been very successful in the treatment of congenital hydrocephalus. Puncture of the ventricles, originally recommended by Hippocrates, and tried again and again, has not generally done any good. The danger of this operation is now much lessened by the use of strict antisepsis, though numerous deaths still occur. Henschen's figures in sixty-three cases give fifteen recoveries, twelve improved, twelve with no change, and twenty-four killed by the operation. Especially dangerous is puncture with after-drainage, as done by Bergmann, Keen, Kocher, and Robson; by it nineteen died out of twenty-two operated upon. The same is probably true of iodine injections, though some good results have been reported.

The surgeons differ widely as to the value of operative treatment and the indications for it. Some think it should only be undertaken in the severest cases; others exclude the mild and severe types, and regard it as a palliative or a curative agent for the other cases. If undertaken, it must be under strict antisepsis; only a small quantity of fluid removed at one time, under control of the pulse, respiration, etc., and compression exerted after it (Huguenin). Henschen thinks it is indicated in progressive increasing intracranial pressure, when life or a special sense is threatened, etc., and contra-indicated in stationary hydrocephalus and bony closure of the cranial sutures. I treated a forty-eight-year-old woman, in whom the operation had been done in childhood, with so much success that we might almost regard it as a complete recovery. Quincke has recommended puncture of the spinal canal, which is not dangerous, but is useless. Aspiration is dangerous. The decrease of pressure should be controlled with a manometer, should be done slowly, and not extend beyond a certain degree. It is more advisable repeatedly to remove small quantities at long intervals.

External revulsion on the cranium has again been revived. It was once so popular that spirit of turpentine was freely applied to the scalp and then set on fire.

Ventricular drainage with horse-hair, subcutaneous division of the spinal dura (Quincke), etc., are some of the recent methods; but we are still in the experimental stage, and are not yet able to establish precise indications for the operative treatment. These surgical procedures can be found more fully discussed in surgical works.

ACQUIRED HYDROCEPHALUS.

Its cause and nature are still obscure. We know that a slight hydrocephalus may exist from birth, unnoticed and latent, until at any time later in life, either spontaneously or from a trauma, insolation, etc., it may exacerbate, and, from a rapid and pronounced increase in ventricular effusion, induce severe symptoms.

But there are numerous cases in which such an origin cannot be assumed. Some types are purely *secondary*, and of not much clinical interest. Congestive hydrocephalus (stauungshydrocephalus) is of this nature, being produced by pressure of a tumor of the posterior cranial fossa upon the greater Galenic vein, thereby causing venous congestion. Compression of the aqueduct of Sylvius may prevent communication between the ventricles, and also between these and the subarachnoid spaces, and in this way produce a hydrocephalus. Cysticerci may also block the communication between the ventricles and cause hydrocephalus; in another, a cicatrix had closed up the fourth ventricle. It is well known that purulent and tubercular meningitis are often accompanied by hydrocephalus. The forms occurring with marasmus, phthisis, nephritis, etc., do not belong here.

External hydrocephalus is mostly of secondary importance; it is the general result of cortical atrophy, be this of senile or other origin. A primary external hydrocephalus also occurs, analogous in symptoms to those of internal hydrocephalus.

We have remaining a form of *idiopathic hydrocephalus*, occurring at any age, though rarely in the aged, surmise as to the origin of which has led to many hypotheses. The most satisfactory is the one which regards it also as secondary, due to a *simple basal meningitis* which has closed the foramen of Magendie, etc. In many cases, however, all signs of meningitis are absent. I described such a case in 1889, and, based upon it, I came to believe in the existence of a *primary idiopathic hydrocephalus* emphasizing particularly its symptomatological connection with cerebral tumor. Annuske had already made a similar observation. In the last few years Quincke has studied this question, and thinks that a *serous meningitis of the ventricles*, a simple serous inflammation of the intracerebral pia (not parasitic, but analogous to serous pleuritis), may be the cause of the internal hydrocephalus in not a few cases, appearing in childhood or later, and running an acute or chronic course. The exudate arises chiefly from the choroid plexus the cortical pia, however, may be the seat of a simple inflammation, and produce an external hydrocephalus. Trauma, mental strain, alcoholism, and the acute infectious diseases have been given as *causes*.

Further observations (Levi, Joel, Kretschmann, and others) have shown that this form of meningitis is also not rare in purulent otitis. It has been referred to toxemia (Seitz).

If it develops acutely it is difficult to distinguish it from purulent meningitis, and less so from the tubercular form. The rise of temperature is, however, absent, or is only slight and inconstant, or the fever which had been present at the commencement soon recedes. The headache is also slighter and the nuchal rigidity not so pronounced. There is no constant—only a periodic—unconsciousness. The visual disturbance is very marked in these cases. The absence of all indications of tuberculosis may guard against some errors. This form of meningitis may also cause spinal symptoms; for instance, Westphal's sign. Goldscheider saw the knee phenomenon return in one case after lumbar puncture. An absolute diagnosis can generally be made only from the further course, as this *acute acquired hydrocephalus*, or acute serous meningitis, ends in complete or partial recovery in some weeks or months. Some cases, however, in which the symptoms (headache, vertigo, vomiting, optic neuritis, cerebellar ataxia, etc.) resemble the chronic form described below, or in which an ocular affection forms the residual symptom of a cerebral disease, become chronic.

Quincke cites several cases in which headache, vomiting, stupor, and optic neuritis form the cardinal symptoms, and in which, after a remittent course of several weeks or months, recovery occurred (under mercurial treatment). An intermittent course is not rare. A fatal issue is more infrequent. The younger the individual is the less easy is it to distinguish this form from that of congenital hydrocephalus.

In another set of cases the course of this disease resembles that of cerebral tumor. Optic neuritis or choked disk and atrophy are almost constant symptoms. The visual disorder repeatedly resembled the type of bitemporal hemianopsia due to the puffed-out floor of the third ventricle, compressing, particularly, the middle piece of the optic chiasm. I found it so reduced in one case that only two thin, thread-like, processes revealed the course of the optic nerves. Intense headache, vomiting, attacks of vertigo and spasms, paralysis of the cranial nerves, exophthalmos, and retardation and acceleration of the pulse, occurred in most cases. A weakness in the extremities was noticed frequently and early, also pain. In many of the cases observed by me there was present a general, rapid, vibrating tremor, which accompanied the active movements. Several times, inclination of the head backward caused very severe pain, vertigo, and vomiting.

None of the symptoms described have anything characteristic about them. There are only two factors that help to make a differential diagnosis. One is, that this hydrocephalus is, in many cases, due to a congenital disposition, which expresses itself in an *abnormal size* and *form of the skull*. The other is the course of the disease, in that *remissions* and *intermissions* of years occur, which is very uncommon in cerebral tumor.

I have described one case of this kind in which the symptoms were succeeded by a period of well-being which lasted for three years, until, during pregnancy, the symptoms again came on.

The variability in intensity of the different symptoms is emphasized by Quincke. The *absence of focal symptoms* is also noteworthy. Even if they appear temporarily they do not gradually progress as in cases of tumor. The *basal cranial nerves* are generally involved from the compression which they undergo.

Further observation must show whether other phenomena, as the early appearance of muscular weakness in the lower extremities, the tremor, or the slight exophthalmos which is often present, can be used for diagnostic purposes. Lumbar puncture and examination of the fluid extracted may help in the diagnosis, the quantity of albumin being increased in tumor and normal in hydrocephalus. This is not very reliable, however.

Some cases after a chronic course exacerbate in an acute manner. This acute stage may, with an incomplete anamnesis, make us think it is an independent disease. Death may occur after months or years.

The difficulties in the way of a diagnosis are then very great,—it is less so in children, in whom the cranium can increase in volume. The closed sutures of adults are also said to have been separated by hydrocephalus, but this is probably a very rare occurrence. The diagnosis of acute serous meningitis is even less firmly established. Lumbar puncture may aid in distinguishing it from other forms of meningitis. It is in this form that a clear fluid, occurring under high pressure (one hundred and fifty to six hundred millimetres water and more), mostly rich in albumin and with a tendency to coagulate (authors differ on this point), and containing neither lymph corpuscles nor micro-organisms, is found. The diagnostic value of this method is, however, considerably decreased by the fact that an increase of the cerebrospinal liquor and increased pressure of it is found in many conditions (chlorosis, uremia, sinus thrombosis, delirium tremens, etc.). Quincke regards it as possible that acute exudates into the ventricles may be a transient phenomenon in periodic headache (migraine) and be somewhat analogous to acute circumscribed edema of the skin.

The prognosis of acquired hydrocephalus is not absolutely bad. Recoveries, improvement, and standstill have been noticed. Life is in danger the more the clinical picture approaches that of cerebral tumor.

Treatment.—In acute cases, which resemble meningitis, the treatment is similar to that of that disease. Quincke recommends a *mercurial* treatment in all cases, whether syphilitic or not. Lumbar puncture is said to be a curative and palliative remedy (Quincke, v. Ziemssen, Lenhartz, Boenninghaus, and others). In the chronic form of serous meningitis, in a case which simulated a cerebellar tumor, I secured a lasting result from spinal puncture, the headache, vomiting, cerebellar ataxia, and paralysis of the ocular muscles completely disappearing, and the visual disorder, which was due to optic neuritis, partly receding. The patient for the last two years has again followed his occupation.

Direct ventricular puncture is preferred by some surgeons, particularly when spinal puncture is unsuccessful. In some cases division of the dura mater of the brain relieved the symptoms. Recovery has been produced especially often in the otitic form of this disease. Von Beck observed the disappearance of coma and Cheyne-Stokes breathing and the return of vision in a few hours from the influence of ventricular puncture.

Revulsive methods are also in place. A *seton* has repeatedly given me good service, while Quincke favors the inunctions of ointments of tartar emetic.

An area of about the size of a dollar upon the closely shaven parietal region is surrounded by a ring of court-plaster, and in this region a mass of ointment, the size of a pea, is to be daily rubbed in with gauze or cotton. After from two to four days an intense inflammation is set up, which leads to necrosis. As soon as the signs of inflammatory swelling have appeared, the inunctions should be discontinued. After a few days edema develops in the neighborhood; at times, also, vomiting, albuminuria, and mild fever—symptoms which disappear with the demarcation. Warm applications help the process, which should be ended in from ten to twelve days. Suppuration should be kept up for from six to eight weeks by unguentum basilicum.

SYPHILITIC DISEASES OF THE BRAIN.

We do not include here those affections which have only an etiologic connection with syphilis and whose anatomic bases do not consist in specific alterations but in simple inflammation and degeneration. This description applies more to the true specific diseases of the brain. These are of various forms. They arise, not including syphilis of the cranial bones, in the majority of cases *from the meninges and the vascular apparatus.* It may be a diffuse, superficial, inflammatory neoplasm, or a circumscribed solitary or multiple tumor formation, or both alterations may be combined.

Syphilomata or *gummatous tumors* are rounded, irregular, knob-like eminences, the size of a hazel-nut or walnut, or larger. Upon cross-section they show a reddish-gray periphery, while in the central parts are found the yellow, dry, viscid, caseous foci. They are often infiltrated by a hard fibrous tissue that may also extend around the circumference in the form of an indurated membrane. The vascular disease, which is either the only affection or exists in conjunction with the above-described gummata and meningitis, is an *arteritis*, involving, particularly, the basal cerebral arteries.

These processes, studied particularly by Heubner, are of various forms (Figs 243 to 245). The gummata and meningitis may extend directly to the arterial wall. An independent endarteritis also often occurs,—a proliferation arising from the cells of the intima which produces a contraction and even an obliteration of the vascular lumen, or is combined with thrombosis. A mesarteritis or periarteritis may also be present in such cases. An arteritis or periarteritis gummosa (Baumgarten, Marchand), with the formation of yellow knobs in the vessel-wall,—the product of a primary round-cell proliferation with caseation of the central part of the external membranes,—have also been described. These different forms may also be combined. These processes differ from ordinary endarteritis deformans in the absence of fatty alterations and calcification, but endarteritis obliterans is by no means exclusively an attribute of lues (Friedländer).

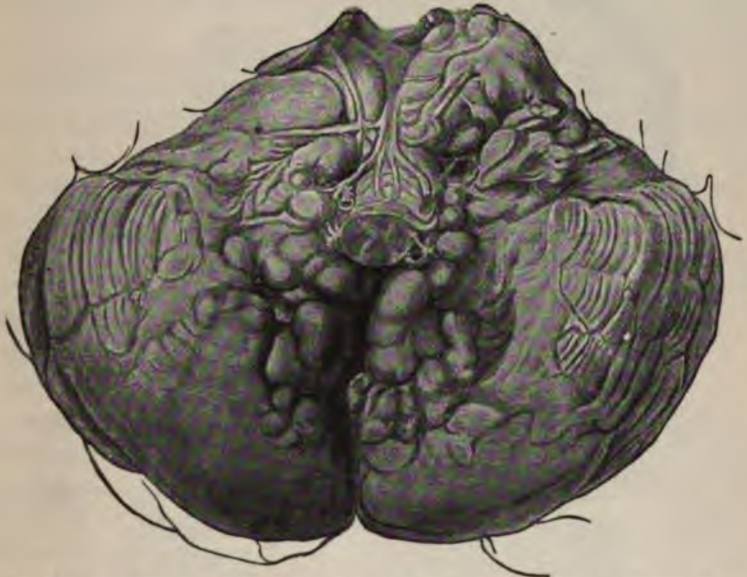
The *chief form* of brain syphilis is a *basal gummatous meningitis.* This generally arises in the subarachnoid tissue of the *region of the chiasma* from the space between the cerebral peduncles, and extends diffusely, though irregularly, more or less over the whole base of the brain. The neoplasm is partly of a gelatinous and partly of a fatty consistency, and often forms, even in large areas, a hard connective-tissue membrane, firmly adherent to the basal parts of the brain. It forces its way into all fissures and depressions, and covers the origins of the cranial nerves like a veil. On closer observation they, particularly the *optic* and *ocular nerves*, are found to be not only surrounded by tumefied tissue, but are also altered, swollen, and upon cross-section appear glossy-gray or a yellowish-white. The cranial nerves which are surrounded by the tumor may, however, appear perfectly normal upon macroscopic inspection. The large arteries at the base of the brain are also involved. Their walls are thickened and adherent to the diseased meninges, and their lumina are contracted (or dilated at places). A circumscribed gumma may exist with the diffuse meningitis, or a basal meningitic process may confine itself to a small area; for instance, the neighborhood of the oculomotor, chiasm, etc.[1] On the other hand, in a few cases, as in one described by Siemerling, extended conglomerations of gummata were found. (Fig. 242.) The rest of the brain may macroscopically show one or more *foci of softening*, especially in the region of the central ganglia or in the pons. *Hemorrhages* are also not rarely found. A diffuse softening over a large area also occurs.

Histologically we find the neoplasm to be composed of a cellular, highly vascular *granulation tissue*, caseated at certain places, fibrinous at others, but never or only rarely purulent. Its character around the blood-vessels (Figs. 244 and 245) and nerve roots (Figs. 246–248) is typical. The round-cell proliferation continues directly along the adventitia of the blood-vessels and also along the *epineurium* of the nerves, especially the *optic* and *oculomotor*. The thickened and infiltrated epineurium sends its processes everywhere between the bundles of nerve-fibres. These processes are the markedly thickened, small-celled, infiltrated, vascular, connective-tissue septa. The nerve-fibres, from the pressure of the swelling and its processes, may partly atrophy, but the nerve itself is swollen *in toto*, even to four or five times its volume, but may in a a later stage appear atrophic. The swelling is due to the new tissue formed in the nerve and to serous imbibition. It is apparent that the superficial layers of the brain itself are involved in the meningeal disease, and gummata may thus extend from the membranes deep into the brain-substance. The same process may develop as a circumscribed or a superficial and diffuse gummatous meningitis upon the convexity of the brain, though not as frequently as upon the base of the brain, and causes disturbances in function of the brain, particularly by spreading more or less deeply into the brain-tissue. This *syphilitic meningo-encephalitis* may lead to softening of an entire lobe or even of a hemisphere.

---

[1] In addition to a diffused and circumscribed extension, a disseminated one, in the form of yellow or gray plaques (meningo-encephalitic foci) or in the form of multiple tumors, is noticed; also miliary gummata are occasionally seen.

FIG. 242.

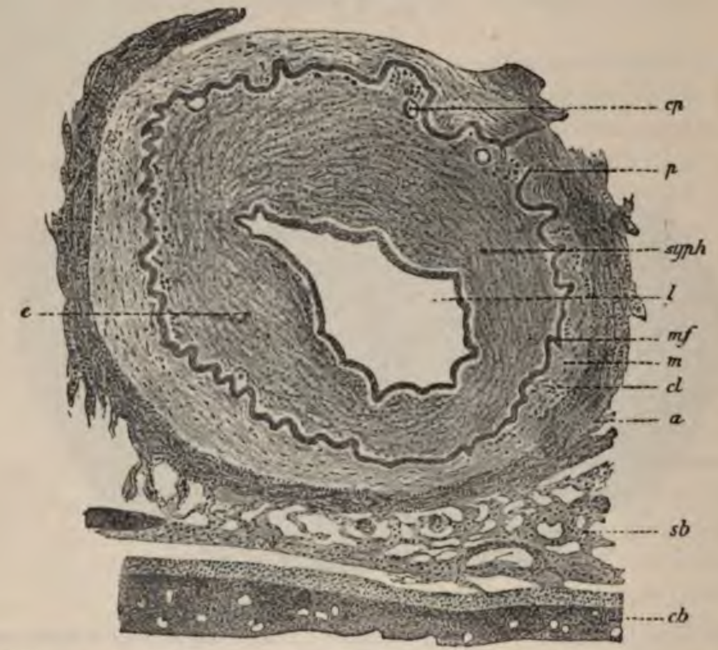


Syphilitic basilar meningitis and multiple gummata on the base of the brain. (Partly after a case of Siemerling's.)

It has been also noticed that the same process may develop simultaneously upon the base and the convexity of the brain. The *multiplicity of the alterations* is to a certain degree characteristic of cerebral syphilis.

Gummata in the interior of the brain are rare. It is noteworthy that *gummatous neuritis of the cranial nerves*, particularly of the optic and oculomotor, may form a primary independent disease, and a *syphilitic arteritis* of several or many cerebral arteries is not infrequently found, without any other discoverable anomalies of the brain or its adnexa.

FIG. 243.



Syphilitic arteritis. Cross-section of a branch of the middle cerebral artery. *syph*, syphilitic neoplasm; *l*, lumen; the syphilitic neoplasm of the intima consists of cell-nests, which are less thick at *e*; *mf*, original fenestrated membrane; *m*, muscular coat; *a*, infiltrated adventitia; *sb*, infiltrated subarachnoidal space; *cb*, upper surface of the brain; *cl*, round cells; *cp*, newly formed capillary; *p*, pigment. (Carmine stain.) (After Heubner.)

An uncommon occurrence is the formation of circumscribed foci of softening, not due to a vascular disease and perhaps of encephalitic origin.

Etiology.—Syphilitic brain diseases are due to *constitutional syphilis*. It is very doubtful that they can be due to a soft chancre, but Hitzig has set up the following hypothesis: "In venereal infection several poisons are inoculated from the beginning or produced in the first stage of the infection. A poison which is present in the primary sore or its resulting conditions leads to secondary and tertiary forms of syphilis. Another poison, which may be present in the same syphilitic ulcer, but which does not need to be present, produces a peculiar diseased alteration of the blood, which after years or decades tends to cause degenerative alterations of the whole nervous system. The same poison may not only be contained in the syphilitic primary affection, but also in the chancre." This hypothesis cannot be applied to brain syphilis in the narrower sense of the word.

The different syphilitic affections of the brain appear most often within one year after the infection; a large percentage occur in the first year or first two years; after ten years the nervous system is rarely involved (Naunyn). In some cases cerebral symptoms have been noticed at the beginning of the secondary stage, several months, or even several weeks, after the infection (Nonne, Saenger, Gilles de la Tourette, etc.).

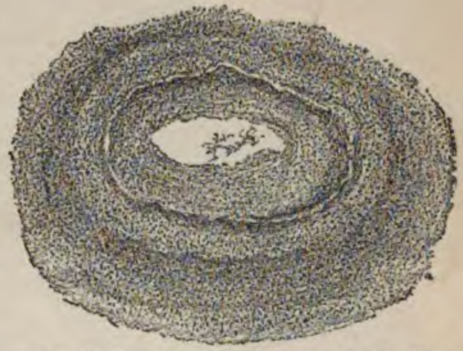
Henschen, in seven hundred and fifty-four syphilitics treated at the hospital of Helsingfors, notes that one hundred and twelve had brain syphilis.

Traumata (cephalic injuries), *mental over-exertion*, *strong emotion*, and *alcoholism* increase the predisposition of the brain to disease. It is often noticed that the first signs of brain syphilis occur soon after some injury to the head.

Symptomatology.—We will commence with the typical form, gummatous basilar meningitis.

The *clinical picture* of this diffuse basal brain syphilis is, notwithstanding its variability, very characteristic. The affected individual becomes sick with general symptoms, of which headache is the most constant and early. It increases in paroxysms to a severe intensity, the exacerbations coming on frequently at night. Vomiting and vertigo frequently occur, and often attacks of *unconsciousness* and generalized convulsions. Some *dementia*, weakness of the memory, and apathy is, as a rule, present. We do not notice, however, the constant progressive stupor which is characteristic of most of the other forms of intracerebral tumors. The patient may be conscious for long periods, but disorders of consciousness occur intercurrently,—a deep stupor lasting several hours or days, or conditions which, on superficial examination, resemble the picture of an injury, drunkenness, or sleep, or severe *attacks of excitation*, paroxysms of confusion and mania. Particularly characteristic is the alternation of delirium with coma; and it is noteworthy that even deep coma will recede and give way to a free sensorium.

FIG. 244.

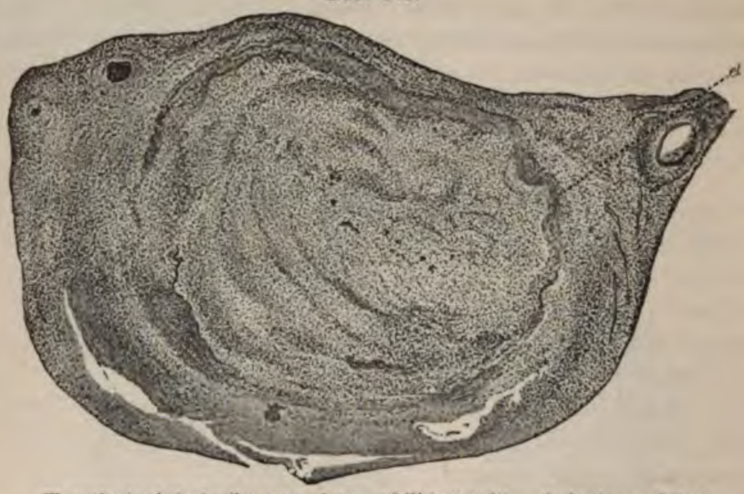


Arteritis from syphilitic basilar meningitis. Cross-section of artery. (Stained with carmine and alum-hemotoxylin.)

In many cases *polydipsia* and *polyuria* are present constantly or only at intervals.

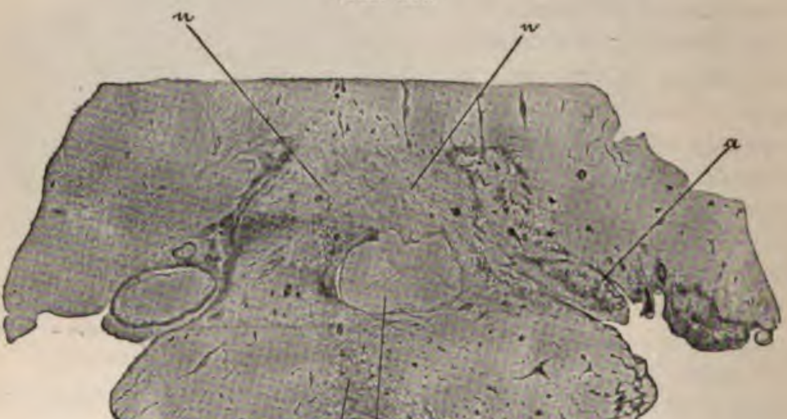
Paralytic symptoms occur at the same time as these general cerebral phenomena, generally following and only occasionally preceding them.

FIG. 245.



Thrombosis of the basilar artery from syphilitic arteritis. *el*, elastic membrane.

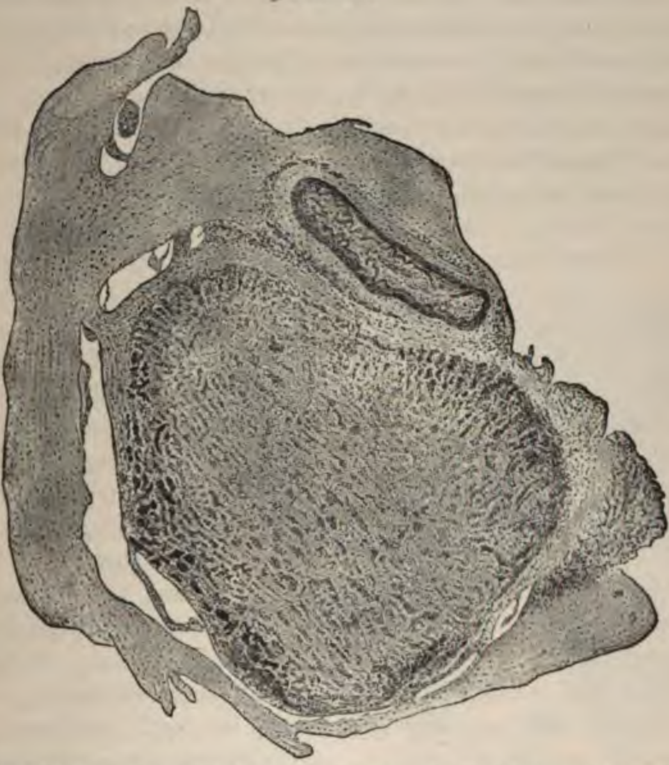
FIG. 246.



Syphilitic neoplasm above the optic chiasm, extending particularly over the middle piece. *n*, neoplasm; *a*, thrombosed artery; *g*, gumma.

In accordance with the anatomic relations, we find that the *optic nerve* and the *motor nerves to the eye* are most often, not rarely exclusively, involved. A paralysis, or paresis of the whole or some branches of the oculomotor, on one side or both, is often seen. The trochlear and abducens are affected less often, the latter generally on only one side. Sometimes the whole oculomotor is attacked at once; in other cases we can see one ocular muscle after another becoming paralyzed. Ptosis is observed very often. The pupillary nerves may be involved by themselves, or after the recession of the cerebral lues pupillary rigidity forms a residual symptom. On the whole, however, a pure ophthalmoplegia interior (and exterior) is *a rare occurrence* in basal syphilis. If both oculomotor nerves are diseased, one side is always more affected than the other, and some of the other cranial nerves are also involved upon this side.

FIG. 247.



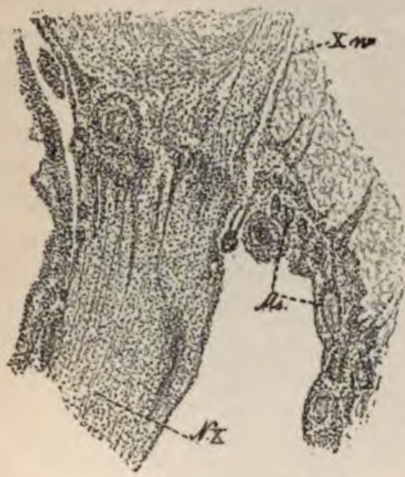
Cross-section of the optic nerve near the bony canal. Pronounced perineuritis, neuritic atrophy, obliteration of the ophthalmic artery. (After Uhthoff.)

Diseases of the optic nerve—Uhthoff observed it in fourteen of seventeen cases of brain syphilis that were autopsied, and found a pathologic ophthalmoscopic picture in forty per cent. of the cases observed clinically—reveal themselves by *ophthalmoscopic alterations* and *disorders in function.* Optic atrophy, typical choked disk, and neuritic atrophy are often found on one or both sides. A simple (descending) atrophy has been detected in many cases. Uhthoff regards it, however, as improbable that a purely primary progressive optic atrophy occurs in cerebral lues.

The ophthalmoscopic examination is often negative or uncertain, while the examination of the vision reveals pronounced disturbances. The conditions found are homonymous hemianopsia or bitemporal hemianopsia; or there develops from a hemianopsia, blindness of one eye with temporal hemianopsia of the other, and finally bilateral amaurosis, which is, however, rarely total and persistent. If the disease-process originates in the optic nerve itself, a concentric or irregular contraction of the visual field, decreased central visual acuity, etc., are present.

The *olfactory* nerve is not rarely embedded in the neoplasm, and unilateral or bilateral *anosmia* occurs. The fifth nerve is more often affected, particularly upon one side, causing irritative symptoms, hyperesthesia especially, but also hypesthesia and anesthesia, and often *neuroparalytic keratitis.* If the process extends farther back, the *facial* and *acoustic* become involved; the facial paralysis has naturally the character of a peripheral one, without, however, an alteration of electrical excitability being always found. A facial diplegia may also result from a basal cerebral syphilis. If the meningitis spreads particularly in the posterior cranial fossa, the *hypoglossal, vago-accessorius,* etc, become involved, producing corresponding paralytic symptoms.

FIG. 248.



Syphilitic meningitis extending to the medulla oblongata and vagus root. (Carmine and alum-hemotoxylin.)

The symptoms do not always denote a continuous extension of the process of such a nature that only cranial nerves arising near each other and taking the same course are affected; on the contrary, the clinical picture often indicates separable foci due partly to the fact that the nerves surrounded by the neoplasm are not equally injured in structure and in function.

The above symptoms indicate a basal disease-process of small foci, extending over a large surface. But this does not reveal its specific nature. Other diseases besides those of a syphilitic nature may, perhaps, have a similar extension, but are not met with very often. Tubercular meningitis is to be excluded by reason of its development, course, and temperature. In syphilitic meningitis the temperature is normal or is only occasionally increased.

But the most important differential point is the peculiar course of brain syphilis, the *variability of the symptoms,* their *coming and going and changing* constantly.

The visual disorders may also undergo this *oscillation* sometimes to an astonishing degree. I found, for instance, in several cases under examination on various days, now a normal visual field, now an irregularly concentric contraction, whose borders varied from day to day, now a pronounced hemianopsia, etc. A *fugitive bitemporal hemianopsia* seemed to me to be particularly characteristic. A temporary and remittent amaurosis, also a remittent choked disk, may occur. The same is true of the paralyses of the ocular muscles. To-day we find a paralysis of the oculomotor; after a few days it may have markedly receded, to return shortly in its full intensity and completeness. I treated a patient in whom ptosis and paralysis of the superior rectus appeared repeatedly in the one eye while I was examining her, and which disappeared in ten minutes to half an hour again while I was still examining her. A coming and going of *reflex iridoplegia* was even noticed in one case. The facial paralysis may repeatedly remit, and, as I have noticed several times jump from one side to the other.

This active variation in the phenomena is explained by the pathologico-anatomic alterations. This granulation-tissue proliferates and dies in rapid succession and constant repetition, and the nerve which is surrounded by it is subjected to a more variable pressure than in any other disease.

The symptoms described above are caused by a meningitis and a neuritis or gummatous perineuritis of the cranial nerves. The phenomena produced by disease of the vascular apparatus, and studied particularly by Heubner and Rumpf, are of great importance. Of these the most constant is the *hemiplegia,* which may occur in every stage, mostly, however, in the advanced. The nature of the process makes it evident that the paralysis does not, as a rule, come on without any premonitory signs. Circulatory disturbances come on first, and afterwards a definite obliteration of the vessel, with resulting softening of the region which is deprived of its nutrition. Slight *apoplectic attacks,* paresthesia, and paresis precede the hemiplegia, until what was at first a *fleeting* hemiplegia becomes permanent in a new attack. The progressive character of the hemiplegia is also characteristic, the leg, then the arm and face, etc., becoming involved in turn. The sensorium may remain intact, or consciousness may be lost. As the arteries are chiefly affected upon the side on which the basilar process is the most advanced, the hemiplegia is generally upon the *opposite* side to that on which the cranial nerves are paralyzed, and the laws of localization for crossed hemiplegia (superior and inferior) must be used cautiously in brain syphilis. For instance, paralysis of the oculomotor of one side and of the extremities of the opposite side are here occasionally caused not by a focus in the cerebral peduncle but by a basal process and its resulting conditions. The hemiplegia may also attack the same side of the body as that on which the cranial nerves are paralyzed.

In the same manner,—*i.e.*, from an endarteritis,—*hemianesthesia*, *aphasia*, and a hemianopsia of central origin occur in some cases.

This severe, often wide-spread, arteritis may also cause a paralysis of the extremities of both sides; it is also not uncommon for the *basilar and vertebral arteries* to be particularly affected, involving the pons and medulla, and thus causing *bulbar symptoms.*

This is the typical picture of a syphilitic basilar meningitis which varies in the most manifold manner according to the parts involved. In some cases, the process is limited to a small area, and, in addition to the headache, the *oculomotor paralysis* or some visual affection is the only objective sign of the disease.

It may run an acute, subacute, or chronic course. It generally develops insidiously, the headache preceding the paralysis by weeks, months, or even years. The further course is also rarely a violent one, but subacute or chronic, and characterized by *repeated remissions and exacerbations*, so that the disease consists of a number of stages of severe sickness, between which complete or partial comfort is experienced. In this manner the disease may last for years.

The prognosis is not unfavorable. Recovery can only be expected so long as merely *specific* tissue alterations exist, and so long as results of compression (atrophy of the cranial nerves) and those of the arterial obliteration (necrobiosis) are not yet present. If, in addition to the general cerebral phenomena, only the signs of a gummatous neuritis are present, complete *recovery* is possible. The ophthalmoscopic examination often shows us whether a defect will remain. An optic neuritis or a papillitis may recede completely, while a pronounced atrophy will not. A cranial nerve which has been paralyzed for years cannot become completely restored to function.

The prognosis as to recovery is made worse with the onset of the hemiplegia. The longer it lasts the more improbable is it that it will disappear. If contractures have come on, a restitution is not to be thought of any longer.

The *danger to life* is always great; it is the greater, the more it affects, directly or through involvement of the basilar and cerebral arteries, the region of the pons and medulla oblongata. *Relapses* are liable to occur. Even with a complete disappearance of the paralytic symptoms a certain mental debility often remains. *Old age* and a *poor condition of nutrition* also cloud the prognosis.

Syphilitic meningitis of the *convexity* of the brain produces pronounced symptoms when it extends over cortical areas which when injured cause disorders of function. Severe, persistent, often locally circumscribed, headache, corresponding to a localized area of sensitiveness on pressure, may be present in all cases, no matter where the disease-process lies. Other symptoms are present, particularly when the motor zone or the speech centre is involved, and these localities are in reality the ones most often attacked. If the motor region is affected the symptoms of cortical epilepsy followed by monoplegia manifest themselves. We have, however, very few points which indicate its specific nature. Here also the step-like course is found. This is, though, of little benefit in the establishing of a diagnosis, as neoplasms of the motor zone cause symptoms which come in attacks *gradatim;* this is the nature of Jacksonian epilepsy. The changeability of the symptoms is, however, more pronounced here than in other diseases of the motor zone, the remissions are generally more complete and of longer duration, and occur even in the paralytic stage. The general symptoms of intracranial pressure, above all the choked disk, may be lacking in specific meningitis until the very end, due to the tendency of the growth to extend superficially. This also has only a limited diagnostic value because (1) glioma of the cortex may exist for a long time without the fundus of the eye revealing anything; (2) syphilitic cortical processes are often combined with a basal growth which can affect the optic nerves directly. Symptoms of irritation—epileptoid attacks, persistent twitchings in certain muscular groups, tremor, contracture, pains in the opposite side of the body, etc.—are, of course, very pronounced.

The symptomatology may indicate spreading of a disease-process over a large superficial area, and in this help in the making of a diagnosis.

I have observed cases of this kind in which a unilateral headache appeared first. After some time spasms in the opposite side of the body manifested themselves which produced a transient monoplegia or hemiplegia, then a persistent one, which varied, however, in intensity; in a new attack, aphasia came on, which also at first appeared periodically until finally it established itself. The rather pronounced impairment of intelligence also denoted an uncircumscribed focus.

Such a development and grouping of the symptoms should always awaken a suspicion of syphilis; the treatment is a valuable aid in determining this question.

In a case described by Wernicke and Friedländer, a bilateral syphiloma of the temporal lobes caused deafness. If the occipital lobe is attacked, hemianopsia and other symptoms may manifest themselves. In an extensive syphilitic meningitis of the convexity the psychic symptoms are very pronounced.

The prognosis is, on the whole, more favorable in syphilitic meningitis of the convexity of the brain than it is in basal syphilis. I have seen many cases of this kind in which recovery occurred under proper treatment. In some of the cases aphasia was present in addition to the headache (an aphasia which had a step-like development); in others, there were cortical epilepsy and monoparesis (with or without sensory disturbances); in others, aphasia and faciobrachial monoparesis. In a number of the cases the paralytic symptoms were conquered while the convulsive attacks persisted, or it was the opposite which occurred.

Isolated gummatous growths are of infrequent occurrence; multiple ones are the rule. The symptomatology, excluding the alternate coming and going of the symptoms, does not differ materially from that of other forms of neoplasms. The symptoms caused by primary syphilitic neuritis require no special description.

The symptomatology of "multiple syphilitic neuritis of the nerve-root" is described by Kahler in the following manner: "Acompanying other signs of cerebral syphilis, perhaps without any, progressive paralyses of various cranial nerves come on insidiously, which are regarded as peripheral. One cranial nerve after another becomes affected by the paralysis in irregular order. A neuralgia, slowly increasing in intensity, appears in an area innervated by different spinal nerves, combined with hyperesthesia, girdle sensations, etc., the results of disease of the posterior spinal roots. Involvement of the anterior roots is revealed by paralysis (peripheral) in the corresponding motor nerves." This description is just a little too schematic.

There are some cases in which the *arterial vascular apparatus* is entirely or principally the part of the body that is affected. Symptoms then only appear when obliteration or, more rarely, rupture of a vessel occurs. According as the thrombus affects this or that branch, we find *hemiplegia*, *hemianesthesia*, *aphasia*, *hemianopsia*, or acute bulbar paralysis. The symptomatology then corresponds in general to that of *encephalomalacia*. Here, also, however, we find indications of the syphilitic nature of the disease. Prodromes, particularly persistent headache and vertigo, often precede the disease; also *apoplectiform* *attacks,* which may cause fleeting *paralyses* in the muscular regions, which later become persistently paralyzed. A condition of *pronounced stupor* and *mental confusion* often follows the apoplectic attacks. *Dementia* occasionally occurs, a symptom which could not be explained by a circumscribed focus. *Pupillary* rigidity may also be found. Kostenitsch found in such a case a small-celled infiltration of the ependymal gray in the region of the oculomotor nucleus with partial atrophy of it.

Vascular cerebral diseases of syphilitic origin have the worst prognosis of all specific diseases of the brain, though antiluetic treatment has been said to cure the vascular affection (Leudet). Phenomena produced by aneurisms of the arteries at the base of the brain and rupture of them are discussed elsewhere.

Symptoms of syphilitic meningomyelitis may combine with those of cerebral syphilis, particularly with those of basal meningitis. This cerebrospinal syphilitic meningitis is one of the commonest syphilitic diseases of the central nervous system. Its clinical symptoms are so easily pictured that they do not need a special description. Occasionally the brain symptoms are so pronounced that the spinal phenomena are entirely obscured; or a loss of one or both knee-reflexes, or an extraordinary weakness in the legs, or a girdle sensation, etc., betrays the spinal process. In other cases the spinal symptoms are pronounced, and every spinal symptom-complex described may combine with every cerebral one. In a large number of cases the spinal disease corresponds to the type of Erb's syphilitic spinal paralysis; and, in combination with the cerebral symptoms, produces a picture similar to that of multiple sclerosis. The special disease may also correspond to Brown-Séquard's unilateral lesion. In another group of cases the symptoms may be similar to those of tabes dorsalis, and in combination with cerebral symptoms produce a picture which I have called syphilitic pseudotabes.

Symptoms of *hereditary syphilitic* brain diseases manifest themselves in the first epoch of life; it is also not uncommon for them not to appear until puberty. They may not come on until the third or even the fourth decade, as I observed in two cases. The symptomatology is, in general, similar to that observed in brain and cerebrospinal diseases due to acquired syphilis, only the inhibition of the mental development is dominant here. Epilepsy is also a prominent symptom. The most important observations have been made by Fournier, Rumpf, Hutchinson, Money, Siemerling, and Boettiger.

The chapters on neurasthenia, hysteria, etc., should be consulted for differential diagnostic points.

Treatment.—In every case of brain syphilis an energetic antisyphilitic treatment is necessary. It is indicated even where there is only a suspicion of luetic trouble. It is best to immediately use mercury, instituting inunctions of three to five grams (forty-five to seventy-five grains) of gray mercurial ointment per day, according to the severity of the case. I prefer treatment by *inunction* to every other method of mercurial treatment.

The ointment should be rubbed into a different part of the body daily. Commence, for instance, with the left arm; select the right arm the next day, then each leg, and finally the back and breast. The ointment must be rubbed in energetically and for a long time (fifteen to twenty minutes). The part rubbed is then bandaged. The patient is to be allowed to walk around when the symptoms are not dangerous, even to go out if the weather be pleasant. He should be given plenty to eat. He should gargle hourly with a four to five per cent. solution of potassium chlorate, and his mouth must be kept very clean, as a severe stomatitis renders interruption of the treatment necessary. After his daily walk, the patient is to be given a warm bath or be sponged off.

Though a definite quantity cannot be made the rule, I regard it as necessary to use, on an average, at least two hundred grams of this drug. If all severe phenomena have then disappeared, I consider it justifiable to interrupt the treatment for some time, to resume it after some weeks or months. If the result is unsatisfactory, the treatment must be continued, though it seems advisable to interrupt it for some time to await the effect, which sometimes is tardy in showing itself.

On the whole, the result of antisyphilitic treatment manifests itself early; as a rule, in from one to two weeks (Naunyn).

In relapses the treatment must be renewed. Many patients find it necessary to undergo this treatment once or twice yearly. Fournier and Neisser are in favor of an intermittent treatment extending over many years. Iodide of potassium in doses of from two to five or ten grams (ʒss–ʒijss) a day—some give much more—often produces remarkable results; we should, however, only depend upon this drug in the mildest cases. The iodide seems to act more rapidly, while mercury seems to produce more lasting results.

Antisyphilitic treatment may fail entirely in many cases. It may be of advantage to combine or follow the mercurial inunctions by a *hydropathic, balneo-, or climatotherapeutic* treatment. The baths of Aachen, Nenndorf, Weilbach, and Toelz have often been of service. In some cases a sojourn in the South, in others a mild cold-water treatment has been beneficial. Neisser and others do not, however, regard it as advisable to prescribe sulphur baths during the inunction cure, as sulphur makes the mercury inefficacious, though the baths may be used if the mercury is given internally. Simple sweat cures, as well as Zittman's treatment, are also beneficial, though hot baths (also cold) should be avoided. The paralysis renders the use of electricity, massage, etc., necessary in the later stages.

To guard against relapses a careful manner of living is to be advised. This includes avoidance of mental and physical strain, emotional excitement, sunstroke, traumata, and particularly excesses in drinking and venery.

Prophylaxis.—The prophylaxis of brain syphilis, to which so many young men fall victims, is similar to that for syphilis in general. It is remarkable with what thoughtlessness even educated young men run the danger of becoming infected. I believe that early instruction and warning may avoid much misfortune in this direction. What religious and ethical education and self-interest cannot do the fear of infection may accomplish when the laity are made acquainted with the full danger of this disease. Much as I am opposed to medicinal education of the people as it is carried on in our dailies and in modern literature, I regard it as necessary for parents and educators to instruct young men on this point. In addition it is certain that thorough treatment of constitutional syphilis is the safest though not an absolute protection from brain syphilis.

PROGRESSIVE PARALYSIS OF THE INSANE (DEMENTIA PARALYTICA).

This disease is unfortunately common in our large cities, and makes victims of many otherwise healthy and sturdy individuals.

Moreau, as early as in 1850, spoke of an increase in this disease, and it is not to be doubted that it has increased considerably in the last decade (Wille, Régis, Snell, Thomsen).

Males from thirty to fifty years of age are particularly affected, but it is not infrequently met with in females. It appears at the end of the second or beginning of the third decade; recent observations show that even childhood is not exempt.

I treated a case of this kind in a fourteen-year-old girl at the commencement of my service at the Charité, and had an opportunity to post it, without, however, daring to make an absolute diagnosis. In the mean time Régis, Wiglesworth, Bury, Strümpell, Clouston, Hüfler, Hirschl, Bresler, and others have published similar cases; and A. Westphal, in one case, by a careful and exact anatomic examination, furnished proof that dementia paralytica was present. This *juvenile paralysis* is relatively frequent in the female sex.

A small percentage of cases occur in old age.

We have become more and more convinced that *syphilis* is the most important cause. Recently this has been shown experimentally in that it was not possible to infect "paretics" by inoculations of chancre poison (Krafft-Ebbing). Though we are not justified in regarding dementia paralytica as a syphilitic brain disease, or in considering syphilitic infection as a necessary preliminary condition, it is certain that syphilitics are more often attacked than are non-syphilitics. In the juvenile cases also acquired or congenital lues was present.

Between the infection and the outbreak of the *paresis*, as this disease is erroneously called in practice, there is an interval of from five to twenty years.

The etiologic importance of *mental strain* and *emotional excitement*, and also of *excesses* of all kinds, should not, however, be undervalued. Individuals who lead an exciting, restless life, whose vocation and position require a varied and disordered thinking and working, accompanied by much anxiety and annoyance, are particularly liable to it. The non-syphilitic, under such conditions, becomes easily affected with neurasthenia; the syphilitic, with dementia paralytica; though we do not say that this psychic factor cannot in itself incite the latter. Venereal and bacchanalian excesses increase the susceptibility to this disease, a number of injurious moments thus acting together (Oebeke, Gudden). Krafft-Ebbing has embraced the etiologically effective factors under the phrase "*civilization and syphilization.*"[1] The factors mentioned above explain why artists, officers, speculators, and merchants represent the majority of those afflicted. Finally, numerous observations have made it certain that *cranial injuries* may be an exciting cause.

Heredity does not play a prominent part in the etiology.

Pathologic Anatomy.—Paralytic dementia is pre-eminently a cerebral disease, though the spinal cord is also generally involved.

In the advanced stage we find upon macroscopic examination an atrophic brain, particularly the cortex. The sulci are deepened, the gyri small. The atrophy involves especially the *frontal* and *parietal* lobes; but other areas, as the insula (island of Reil) and a part of the temporal lobe, are also affected.

The *pia mater* is frequently adherent to the brain, so that it cannot be stripped off without some of the cortical substance clinging to it. It may later, however, be easily peeled off. It is often thickened and clouded. A slight or pronounced *external hydrocephalus* often results from the cortical atrophy; the contents of the dilated ventricle become increased, and its walls are covered with granulations (ependymitis granularis). The characteristic alterations of internal hemorrhagic pachymeningitis are often found on the dura. The weight of the brain is generally decreased from about fourteen hundred to one thousand or nine hundred grams, the loss being chiefly in the frontal and parietal lobes. Upon cross-section the cortical gray matter often appears decreased, even to one-third of its normal breadth.

---

[1] Recent observations in America have tended to show a remarkable increase of paralytic dementia in the negro, a race in which it was formerly unknown.

The *microscopic* examination has not as yet given any definite results. It is certain that the nervous elements undergo a partial atrophy; but whether the atrophy of these is the primary lesion, or whether the process starts in the interstitial tissue (vessels, neuroglia), is a question not yet answered.

Various alterations in the ganglion cells have been discovered by Binswanger, Meschede, Mendel, Mierzejewsky, Lubimoff, Tigges, Alzheimer, Nissl, Colella, and others. The interstitial tissue and the blood-vessels are also affected. Increase in the number of nuclei and fibres of the neuroglia, proliferation of spider cells, hyaline and colloid degeneration of the vessel walls, sclerosis of the blood-vessels, dilatation and displacement of the lymph tracts, etc., have been found (Magnan, Mendel, Schüle, v. Recklinghausen, Reynaud, Joffroy, and others).

It has been decided that the process is the result of a neuroparalytic hyperemia, which induces transudation with secondary lymph congestion. This latter then causes the degeneration of the nervous elements. Alzheimer, using Weigert's new method, found glia proliferations over the whole cortex.

Examinations by means of Golgi's, Marchi's, and Nissl's methods have not revealed the nature of the process, though they have called more attention to its parenchymatous nature.

Tuczek has discovered an important fact,—namely, the *degeneration* of the fine medullated fibres (the *tangential fibres*) in the upper layer of the cortex, particularly in the frontal lobe (gyrus rectus) and in the insular cortex.

In a few cases focal softening or pronounced local degeneration has been found (Rosenthal, Ascher, Lissauer, Boedeker). On the whole, Wernicke's definition seems to be adequate in all cases: he says that paralytic dementia is a chronic progressive disease of the cortical substance of the cerebral hemispheres, which causes atrophy of the nervous elements in it.

Degenerative alterations have also been detected in the deeper layers, in the basal ganglia, and in the nuclei of the brain-stem. In the *spinal cord* (Westphal) we find the crossed pyramidal tracts or the posterior tracts diseased, or both together.

Westphal regards the degeneration of the pyramidal tracts as a primary one; Boedeker and Juliusberger found it in one case to be secondary.

Gray degeneration of the optic nerves, more rarely an analogous affection of the other cranial nerves, has been noticed. Recent observations (Hoche and others) indicate that the peripheral nerves may also degenerate in this disease.

Symptomatology.—The cardinal symptoms of this disease are a *progressive enfeeblement of the mental faculties, speech disturbances, paralytic attacks*, and *Argyll-Robertson pupils.*

The mental symptoms are the most important. They appear so prominently that the disease is rightly regarded as a *psychosis.* The basis of the psychic disturbance is the progressive dementia. Decreased intelligence is already observable in the first stage. The finer mental emotions, the ethical concepts and sensations, are first diminished. The individual uses expressions and commits acts which are in the beginning only recognized as peculiar by the close observer and the near relatives. Previously sensible and conscientious, he becomes frivolous and fickle; formerly considerate, he becomes indelicate and vulgar, appears cynical, grossly offends the proprieties, etc. The higher psychic and emotional centres are first disordered. The patient loses interest in his family and becomes cold and heartless. He soon becomes incapable of real mental work and neglects his business duties more and more. The enfeeblement of the mind shows progression when the *critical faculties* are so diminished that the previously upright man commits acts which place him in conflict with the law. As a rule, these indicate not only an *ethical* but also an *intellectual* defect; though wealthy, the patient steals an almost worthless object; though married happily, he runs after a strumpet, etc. If the prodromes pass unrecognized, the disease may suddenly be noticed from the bizarre and surprising acts which the patient commits. A paretic defecated on a public street in daylight; another poured champagne into women's pockets; a rich man stole a sausage in passing a meat-shop, etc.

His memory fails him markedly, often at the beginning of the disease, particularly for recent events. The ability to receive and fix new impressions and to concentrate his attention is mostly impaired, while the memory for old events may be retained for a long time. An abnormal *irritability* also occurs. Slight matters make him exceedingly excited; he is easily angered; his anger does not, however, last long.

If it is not possible to secure data from friends of the patient's family and social life, sufficient to reveal these defects, a cursory examination will suffice to obtain the required information. This should embrace the social standing, occupation, education, and previous pleasures of the patient. Let him give a short description of his life; inquire concerning the occurrences of the last few days; ask him some questions pertaining to his occupation. Loss of mental computation is particularly pronounced. Mental computation cannot be expected of every one, though it is more than suspicious if a bookkeeper, banker, etc., cannot multiply two numbers of two figures in his head; failure of a day laborer to multiply single figures need not necessarily denote anything. The previous capacity must be known. In advanced cases the time of the year, his age, etc., may no longer be known by the patient.

In examinations where I am desirous of rapidly reaching my goal, or where I wish to suddenly show my listeners the mental enfeeblement, I generally ask the following questions: "What year is it?" If the patient answers correctly, say 1900, I ask what year it was five or seven years ago, and in advanced cases almost always receive a false reply. Or I ask, "How much is seven times nine?" The patient answers correctly, "Sixty-three;" and then I ask, "How much is nine times seven?" and my patient thinks a long time, to give finally a correct or false answer. The mental weakness also reveals itself early in the *facial expression.* In no other disease can we so easily establish a diagnosis at the first glance.

The dementia increases rapidly or slowly to end in complete mental darkness, in deep idiocy. In a certain percentage—according to recent observations, in a considerable number—of cases simple *apathetic dementia* is the only sign of mental disease noticed. It is generally characterized also by *hallucinations* and delusions and a *pronounced alteration of the emotions.*

The delusions are generally of a grandiose character combined with ideomotor excitement. The patient thinks he is a great artist or a discoverer, or a detective who daily and hourly exposes something new and horrible. He is king, God, possesses uncountable millions, the whole world belongs to him. These grandiose ideas are, as a rule, not permanent, but are easily displaced by others; nor is the patient able to give the slightest logical basis for them.

Hypochondriac ideas also often govern the thoughts of the patient and, particularly when they appear at the onset of the disease, produce deep depression. These crass and absurd notions early reveal the mental debility which is present. The patient imagines that he has no stomach or intestines; that food passes to his brain; that he cannot swallow; that his head is too large or too small, etc. These ideas may be so masterful that food is refused, and enfeeblement and even starvation may result. Generally, however, they do not last long, but give way to grandiose ideas. In the beginning of the disease the certain knowledge of the commencing malady may induce *deep melancholia.*

Ideomotor excitement often appears. If it manifests itself in the initial stage it may resemble *mania.* The patient executes all kinds of movements, mutters continually and with great volubility, strikes at everything around him, injures himself and others, screams, scolds, yells, and no sentence is completed, but only senseless syllables or disjointed words are spoken. He is constantly making grimaces, refuses nourishment, and rapidly emaciates. Some are in constant *terror* of some impending trouble. The farther advanced the disease is the more evident becomes the mental debility.

Somatic symptoms are, generally, early combined with the psychic phenomena. The *reflex pupillary rigidity* deserves first mention, because it is not only a frequent but also an early symptom. It may even occur years before the disease, and as a *mene tekel* reveal the fate of the individual. The pupils are generally contracted, though mydriasis also occurs in conjunction with the immobile pupil.

Westphal's students (Moeli, Thomsen, Siemerling, Wollenberg, Gudden) have studied the condition of the pupils in a large number of paretics, and found pupillary rigidity in from fifty to sixty per cent. Siemerling makes it sixty-eight per cent. In some cases it preceded all other symptoms by from five to ten years. The pupils are also often angular and irregular.

A pronounced pupillary difference and also hippus may be early symptoms. Paralyses of the external ocular muscles and of accommodation are rarely observed in the initial stage. Hemicrania with scotoma scintillans has often been noticed in the prodromal stage.

An equally important and almost pathognomonic symptom is the paretic speech. It is particularly characterized by a syllabic stumbling or drawling, by elision of syllables, by the trembling and quaking of the lips in articulation, by movements in facial muscles which ordinarily are not exercised in articulation, and often by a slight nasal tone. The speech disturbance is generally noticeable in ordinary conversation. To bring it out prominently the patient should be asked to repeat words or word-combinations which are difficult to pronounce, as artillery brigade, truly rural, blue bottle of blackberry brandy, extraterritorial principle, etc. It is also advisable to have the words repeated several times to see whether the disturbance increases thereby. In reading aloud the articulatory disturbance also reveals itself. That diction and the flow of words likewise suffer from the dementia is apparent.

Later on the handwriting becomes affected: it becomes trembling; the letters very unequal; some strokes are too thick, others too long; letters, syllables, or words are omitted or repeated; syllables are displaced, etc., until finally the contents become senseless. A peculiar form of paralexia has also been described.

Milder grades of these disturbances of writing occur in normal persons, so that the writing should be compared with that of the patient's previous life.

The other movements of the arm also become awkward, though the strength is not markedly impaired. The feeling of uncertainty and of heaviness involves the legs also, the gait becoming shuffling and heavy. The impairment in motility does not, however, except in the attacks to be described below, increase to paralysis, though paralyses of a peripheral type, particularly peroneal paralysis, have been observed several times (Moeli, Pick).

A *tremor* is not infrequently observed, confined to certain muscular regions, or to one side, or spread over the whole body. The labial and glossal muscles are especially affected. Fibrillary tremor and quaking of the lips are noticed, particularly in attempts to speak or in extending the tongue. The latter movement is executed very characteristically: the tongue is jerked out, the mouth being open to its full extent, or is repeatedly stretched out and retracted. The tremor in the extremities is now slight, now pronounced; it is an inconstant, unequal tremor with moderate or great frequency of the oscillations, which accompanies all movements, but is also present when the parts are at rest. *Tabetic* symptoms are often combined with those of paralytic dementia. We do not need to refer again to the Argyll-Robertson pupil. *Optic atrophy* is sometimes present, and may even precede the psychic alteration by years. Westphal's sign, mild ataxia, Romberg's symptoms, bladder disturbances, lancinating pains, etc., are not rarely present. Dementia paralytica may even accompany a completely developed tabes.

More often, however, we find symptoms of involvement of the lateral or of the combined lateral and posterior tracts present even in the first stage. The deep reflexes are pronouncedly exaggerated and the motor strength is somewhat diminished. The trouble rarely increases to spastic paraparesis or to paraplegia. In a few cases severe spinal symptoms of this nature precede the mental symptoms.

We have yet to describe the periodic *paralytic attacks* which generally occur at some time in its course. These attacks are of various kinds. We can distinguish an *apoplectic* and an *epileptic* form. They last, as a rule, from one or several minutes to half an hour or more. The former consists in a temporary lapse or loss of consciousness, followed by hemiplegia, monoplegia, or aphasia. This paralysis lasts only a few hours or days to again disappear. Persistent paralyses of this nature are the exception (Ascher, Rosenthal). Sometimes it is only a slight syncope, followed by a fleeting paralysis, or only an attack of vertigo. Disturbances of consciousness may, however, be entirely absent, so that a transitory paralysis or an aphasia of only a few minutes' duration represents the attack. These critical episodes tend to repeat themselves, and after each attack the patient's condition becomes worse, particularly the mental state and the speech.

The epileptiform attacks represent either *petit mal* or genuine epileptic attacks. In some cases these may precede the outbreak of the disease. More often we find cortical epileptic attacks, either motor or sensory, unilateral twitchings, and more rarely paresthesia with or without unconsciousness. These attacks may follow each other in a series and produce a true status epilepticus. Rhythmical twitchings, isochronous to the pulse, have been described by Kemmler. Some speak of paralytic attacks of a purely psychic nature and include under them conditions of confusion and excitement of sudden onset and rapid disappearance.

The paralytic attacks occasionally cause a *visual disturbance* which corresponds to the type of psychic blindness (Fürstner, Stenger). It generally disappears soon.

The cause of these attacks is not known. We assume that circulatory disorders, small hemorrhages, local edema, or a local excess of the anatomic process at the base of the disease itself (encephalitis?) bring them forth. Recently minute alterations have been detected by Marchi's method. Some authors, as Pierret, have sought to refer these seizures to auto-intoxication. It is possible that peripheral irritation (distended bladder, etc.) may incite them.

The temperature is increased during the attacks; an increase of body heat is sometimes noticed at other times.

Vasomotor and trophic disturbances (perforating ulcer, spontaneous fracture, bed-sores, arthropathy) are not infrequently observed.

Course of the Disease.—The disease develops insidiously, as a rule; the further course is also a protracted one. The symptoms may, however, suddenly exacerbate, particularly after a paralytic attack. *Remissions* are not uncommon; the symptoms may completely recede, so that the laity regard the patient as being completely cured and able to resume his occupation. Almost always, however, is this apparent recovery deceitful; after a few months or a half-year, rarely longer, a relapse occurs. Two or three years after the beginning of the disease, the mental weakness has generally progressed to idiocy, and speech has become so bad that the patient can hardly make himself understood. Finally speech becomes entirely lost. The facial expression betokens complete loss of intelligence. Walking becomes more and more difficult, finally impossible. The urine and feces are passed in bed, the patient smears himself with the excrement, bed-sores may develop, etc.

Death results in from two to three years. There is an acute form in which death comes on in a few weeks or months. On the other hand, the disease may last many years, even as long as a decade. It runs a less rapid course in women. The form with dementia and the tabetic form seem to last longer than those combined with pronounced depression and violent exaltation. Death results from inanition, inhalation pneumonia, decubitus, cystitis, or from any intercurrent malady. The patient rarely dies in a paralytic attack or in status epilepticus.

The prognosis is bad. Every experienced psychiater knows of one or more cases in which he has diagnosed paresis, and in which recovery ensued ; but they compose a decreasing minority, and it is probable that an error in diagnosis had been made in every such case. Krafft-Ebbing, among two thousand five hundred cases, did not observe one recover, but cited an observation of Svetlin which shows without doubt the possibility of recovery taking place.

Diagnosis.—Paralytic dementia is a disease which every practising physician should be able to recognize in order to avoid, by a diagnosis at the right time, any misfortune to the patient's family. It is not always easily established. The *initial stage* offers many difficulties. The early symptoms may resemble those of *neurasthenia*. I have seen several cases in which I diagnosed neurasthenia at first, to find after a year or more that paresis was present. The opposite occurs much more rarely. In such cases, first search for symptoms which are not peculiar to neurasthenia, particularly Argyll-Robertson's pupil or a very sluggish pupil, Westphal's symptom, and paretic speech and gait. It should not be forgotten that neurasthenics, in conditions of excitation or after a long siege of insomnia, may occasionally show a similar speech disorder. But the phenomenon is here transitory and inconstant, and disappears after several repetitions of the paradigm. If these symptoms are absent, a careful examination of the mental state will generally reveal the trouble. Neurasthenics complain also of weakness of the memory, but it is generally based upon a false, morbid self-observation. Whenever the characteristic alterations of the intellect described above are found, neurasthenia is not present, but probably beginning paralytic dementia.

A neurasthenic will give a clear, detailed account of his trouble, showing how sharply he observes himself, how he detects every sensation, and how accurately he is aware of every pathologic occurrence. For this reason, whenever a patient fears that he has paresis, the chances are against his having that disease. In the description of a paretic, apathy, indolence, and mental decay are generally noticed early. There are cases of this kind, however, in which the patient is heavily burdened by his symptoms ; they dominate him and produce a deep and persistent depression rarely noticed in neurasthenia.

Compulsive and fixed ideas speak *in dubio* for neurasthenia.

Much difficulty may be furnished by the not rare combination of tion represents the attack. These critical episodes tend to repeat themselves, and after each attack the patient's condition becomes worse, particularly the mental state and the speech.

The epileptiform attacks represent either *petit mal* or genuine epileptic attacks. In some cases these may precede the outbreak of the disease. More often we find cortical epileptic attacks, either motor or sensory, unilateral twitchings, and more rarely paresthesia with or without unconsciousness. These attacks may follow each other in a series and produce a true status epilepticus. Rhythmical twitchings, isochronous to the pulse, have been described by Kemmler. Some speak of paralytic attacks of a purely psychic nature and include under them conditions of confusion and excitement of sudden onset and rapid disappearance.

The paralytic attacks occasionally cause a *visual disturbance* which corresponds to the type of psychic blindness (Fürstner, Stenger). It generally disappears soon.

The cause of these attacks is not known. We assume that circulatory disorders, small hemorrhages, local edema, or a local excess of the anatomic process at the base of the disease itself (encephalitis?) bring them forth. Recently minute alterations have been detected by Marchi's method. Some authors, as Pierret, have sought to refer these seizures to auto-intoxication. It is possible that peripheral irritation (distended bladder, etc.) may incite them.

The temperature is increased during the attacks; an increase of body heat is sometimes noticed at other times.

Vasomotor and trophic disturbances (perforating ulcer, spontaneous fracture, bed-sores, arthropathy) are not infrequently observed.

Course of the Disease.—The disease develops insidiously, as a rule; the further course is also a protracted one. The symptoms may, however, suddenly exacerbate, particularly after a paralytic attack. *Remissions* are not uncommon; the symptoms may completely recede, so that the laity regard the patient as being completely cured and able to resume his occupation. Almost always, however, is this apparent recovery deceitful; after a few months or a half-year, rarely longer, a relapse occurs. Two or three years after the beginning of the disease, the mental weakness has generally progressed to idiocy, and speech has become so bad that the patient can hardly make himself understood. Finally speech becomes entirely lost. The facial expression betokens complete loss of intelligence. Walking becomes more and more difficult, finally impossible. The urine and feces are passed in bed, the patient smears himself with the excrement, bed-sores may develop, etc.

Death results in from two to three years. There is an acute form in is of another type. *Senile dementia* which, as a rule, appears only after the sixtieth year, is similar to this form. Alzheimer and Binswanger have made a special study of the differential diagnosis of these conditions.

In senile dementia the physical signs of progressive paralysis of the insane are generally lacking; in the aged the pupils are narrow and sluggish, but pupillary immobility is very rare. The syllabic stammering or stumbling is also not present.

Binswanger described also a chronic progressive subcortical encephalitis, in which the white matter of the posterior part of the brain is said to atrophy; the process, however, appears to be only of anatomic interest.

Alcoholism may produce symptoms which are to some extent similar to those of paralytic dementia. To these belong the tremor, the motor restlessness, and the speech disturbance. Whenever alcoholism produces these symptoms, it evokes delirium tremens at the same time, and this condition is so dissimilar to the mental state in paralytic dementia that a differentiation is easily made.

The psychosis occurring with alcoholic neuritis also presents the signs of mental weakness, but is easily differentiated from that of general paresis by the numerous illusions and hallucinations and their typical nature, and by other signs of alcoholism. The Argyll-Robertson pupil is also always or nearly always absent, and the sluggish reaction disappears upon abstinence. There is, therefore, hardly any justification for speaking of an alcoholic pseudoparesis. Bruns described a case in which *uremia* simulated the picture of general paresis. Landenheimer described a diabetic pseudoparesis. In liver diseases also a symptom-complex is said to develop, which presents a superficial similarity to that of general paresis (Joffroy). Chronic lead intoxication may produce a symptom-complex which is very similar to that of paralytic dementia, but which occasionally terminates in recovery (Régis, Ball, myself). Similar conditions may also result from bromine and trional intoxication, which are, however, easily recognized from the etiology. It may be more difficult to distinguish certain forms of morphinomania from paralytic dementia.

It is not in place here to differentiate paralytic dementia from mania, melancholia, etc. We wish it only to be remembered that when any of these psychoses, particularly mania, appear in a previously healthy person, general paresis must be thought of. Generally, the physical symptoms or the mental weakness enable us definitely to recognize this disease. A melancholia or severe hypochondriasis developing, to a certain extent abruptly, in a mature man previously sound and normal, is suspicious.

Treatment.—It is hardly possible for the physician to cure this disease, but, by proper and seasonable measures, he may be able to avoid much harm and establish the most favorable conditions for improvement.

tabes dorsalis and neurasthenia. In such cases, a diagnosis of paralytic dementia should be made only when pronounced mental disturbances or paralytic symptoms foreign to tabes (paretic speech) are present.

The paralytic attacks may, in doubtful cases, be of much diagnostic aid; naturally only when they are *typical seizures*, unilateral convulsions, vertiginous attacks, and temporary inhibition of speech occurring also in hysteria and hystero-neurasthenia. It should further be remembered that conditions very similar to paralytic attacks may occur in hemicrania.

Of other diseases of the nervous system which may be confused with paresis, cerebral lues and multiple sclerosis are the most important.

Gummatous meningitis may, if it involves the region of the speech centre and the motor zone, cause attacks of temporary paralysis of speech and of the extremities, and also produce convulsive seizures very similar to those of general paresis. Headache is, however, generally found; a local sensitiveness of the cranium to pressure is often noticed; and even if the paralytic symptoms tend to disappear at first, some paresis remains in the interparoxysmal time,—in short, the whole course denotes a focal disease. Optic neuritis is indicative of specific disease, while a simple optic atrophy *in dubio* speaks for paralytic dementia. The treatment may also clear up the difficulty, as an antisyphilitic treatment, as a rule, does good only in true lues, being without any effect in progressive paralysis. A spontaneous remission may come on during treatment and make the differentiation more difficult. In cerebral lues, paroxysmal attacks of stupor constitute the important element of mental disturbance; in general paresis, the dementia. I do not regard it as impossible that paralytic dementia may develop from a true cerebral lues. There is a form of diffuse syphilitic meningo-encephalitis, the differentiation of which from paralytic dementia is not always possible. French authors, particularly, speak of a syphilitic pseudoparesis; they claim that there are countless intermediary conditions between it and true general paresis.

A part of the criteria given above apply also in the differentiation of cerebral tumor from paralytic dementia.

Consult page 214 for a differentiation between general paresis and multiple sclerosis. Schultze and Zacher believe that there is a mixed form of the latter and general paresis; this must, however, be a rare occurrence.

There is a diffuse cerebral disease due to *arteriosclerosis*, an atrophy which resembles paralytic dementia in many respects; but in it we almost always find focal symptoms (hemiplegia, dysarthria, dysphagia, etc.) in addition to the mental defects. Further, the dementia seldom becomes as pronounced as in general paresis, and the speech disturbance is of another type. *Senile dementia* which, as a rule, appears only after the sixtieth year, is similar to this form. Alzheimer and Binswanger have made a special study of the differential diagnosis of these conditions.

In senile dementia the physical signs of progressive paralysis of the insane are generally lacking; in the aged the pupils are narrow and sluggish, but pupillary immobility is very rare. The syllabic stammering or stumbling is also not present.

Binswanger described also a chronic progressive subcortical encephalitis, in which the white matter of the posterior part of the brain is said to atrophy; the process, however, appears to be only of anatomic interest.

Alcoholism may produce symptoms which are to some extent similar to those of paralytic dementia. To these belong the tremor, the motor restlessness, and the speech disturbance. Whenever alcoholism produces these symptoms, it evokes delirium tremens at the same time, and this condition is so dissimilar to the mental state in paralytic dementia that a differentiation is easily made.

The psychosis occurring with alcoholic neuritis also presents the signs of mental weakness, but is easily differentiated from that of general paresis by the numerous illusions and hallucinations and their typical nature, and by other signs of alcoholism. The Argyll-Robertson pupil is also always or nearly always absent, and the sluggish reaction disappears upon abstinence. There is, therefore, hardly any justification for speaking of an alcoholic pseudoparesis. Bruns described a case in which *uremia* simulated the picture of general paresis. Landenheimer described a diabetic pseudoparesis. In liver diseases also a symptom-complex is said to develop, which presents a superficial similarity to that of general paresis (Joffroy). Chronic lead intoxication may produce a symptom-complex which is very similar to that of paralytic dementia, but which occasionally terminates in recovery (Régis, Ball, myself). Similar conditions may also result from bromine and trional intoxication, which are, however, easily recognized from the etiology. It may be more difficult to distinguish certain forms of morphinomania from paralytic dementia.

It is not in place here to differentiate paralytic dementia from mania, melancholia, etc. We wish it only to be remembered that when any of these psychoses, particularly mania, appear in a previously healthy person, general paresis must be thought of. Generally, the physical symptoms or the mental weakness enable us definitely to recognize this disease. A melancholia or severe hypochondriasis developing, to a certain extent abruptly, in a mature man previously sound and normal, is suspicious.

Treatment.—It is hardly possible for the physician to cure this disease, but, by proper and seasonable measures, he may be able to avoid much harm and establish the most favorable conditions for improvement.

Though there is no border line between the pons and the medulla oblongata, it is advisable from a nosologic view to draw such a line, and to consider as part of the pons that section which upon the basal surface is covered by the so-called superficial pontine strata (stratum superficiale pontis).

A glance at Figs. 206–212 shows that the pyramidal tracts are found in the basal section (Fussabschnitt) of the pons, separated in the proximal parts by the deeper transverse fibres of the pons into several fasciculi, while they gather themselves more and more into a compact bundle in the distal part.

Above these tracts in the *tegmentum*, we meet with the *lemniscus*, which contains a large part of the sensory conducting tracts. The alterations in position and form can be seen from the illustrations. It is to be particularly noted how in the deeper distal sections of the pons both fillets lie close to each other and right on the raphe, so that they may be injured by a comparatively small lesion, while cerebrad they separate more from each other. That the reticular field, lying above the lemniscus or fillet, contains a part of the sensory conducting paths can hardly be doubted.

The *spinal trigeminal* root can be traced throughout the pons, lying in the lateral area. A lesion to injure the roots of both sides must therefore cover a large extent.

In the lower sections of the pons we find the *acoustic nuclei*, under the floor of the fourth ventricle; in the lateral regions, the *facial nucleus*. Above these come the *abducens nucleus* and the abducens root. Farther up we reach the area in which, laterad, in the corner under the floor of the fourth ventricle, are the *motor* and *sensory* trigeminal nuclei. The fourth ventricle now becomes roofed over by the medullary velum, and the walls are formed by the upper cerebellar peduncle. At this height the *trochlear root* appears, and soon after it the trochlear nucleus.

The fourth ventricle passes into the aqueduct of Sylvius; in this region the upper cerebellar peduncle is pressed inward and downward, and the posterior quadrigemina lift themselves over the aqueduct. We may regard this region as the upper border of the pons.

Diseases of the pons Varolii, for the most part, cannot be separated from those of the medulla oblongata. It appears advisable, however, to discuss separately the *symptomatology of focal diseases* which develop in the pons.

It is apparent that the phenomena are largely dependent upon the *size* and *position* of the lesion. Small pontine lesions may not excite any symptoms, particularly when they have their seat in the gray pontine nuclei or in the transverse fibres. A *nidus*, which injures the *pyramidal tract* of one side more or less completely, produces *hemiplegia*,—a hemiplegia of the ordinary type if it affects the pyramidal tract in the proximal section of the pons. If it injures the tract just above the facial nucleus, facial paralysis is absent, because the central facial fibres have already crossed the median line to reach the nucleus of the contralateral side.

If the lesion is so extensive as to involve the neighboring facial nucleus or its roots, *alternating hemiplegia* results. The facial paralysis is here a *degenerative* one (or it may be) when the nucleus itself or the exogenous roots are injured; it is a simple, non-atrophic one when the central facial root-fibres coming from the other side to the nucleus are injured. The *abducens* may be easily affected with the facial tract. We would then have an alternating or crossed hemiplegia of the sixth nerve and of the opposite half of the body.

Those fibres which pass from the brain to the other nerve nuclei lying in the medulla oblongata also traverse the pons. Pontine lesions may, therefore, also impair the movements of the *tongue*, *pharynx*, *larynx*, etc. These symptoms appear most prominently when we are dealing with a lesion embracing both pontine halves. This may cause *bilateral paralysis of the extremities*, and entail all the symptoms of *acute bulbar paralysis*. It appears that the lesion needs only slightly to overstep the median line to injure the supranuclear tracts of the motor cranial nerves upon both sides.

Bladder disturbances often occur in pontine lesions, particularly bilateral ones.

Injury to the *fillet* of one side produces, notwithstanding the opinion expressed by some authors, sensory disturbances of the opposite side of the body. This is, however, probably never complete, because the sensory fibres partly traverse other paths (page 405).

A lesion which injures a large part of the reticular field may also produce sensory disturbances. The separation of the tracts for the different qualities of sensation is not as yet absolutely possible (Moeli and Marinesco). That pontine diseases may also cause *ataxia* has been established by numerous observations. It has not been proven that the paths for it are entirely or even partly the same as those for sensation, but ataxia has repeatedly been observed in the limbs of the opposite side in diseases which separate the fillet fibres. An affection of the brachium conjunctivum and red nucleus probably produces "cerebellar ataxia." Unilateral pontine lesions which have their seat in the neighborhood of the abducens nucleus may manifest themselves in an *associated paralysis of the ocular muscles*. The eyeball cannot be moved towards the side on which the lesion occurs, and deviates towards the other side. Stimulation of this centre seems to be capable of causing nystagmus.

The symptoms caused by a lesion of the spinal trigeminal root and of the trigeminal nucleus need no special consideration.

Recent observations (Eisenlohr, Bregmann, Wallenberg) indicate that the distal sections of the root correspond to the first branch, etc. (See page 419.)

Lesions in the upper part of the pons which affect also the quadrigeminal region evoke *paralysis of the ocular muscles*, particularly difficulty in moving the eye downward and upward (Wernicke, Nothnagel), *pupillary defects*, especially mydriasis, sluggish reaction or pupillary immobility (Eisenlohr), *disturbances of co-ordination, decreased central visual acuity* (pregeminum, quadrigeminal arm or external geniculate body), and, perhaps, *impaired hearing or deafness* (postgeminum).

Monakow claims that a lesion of the entire pregeminum only produces a slight decrease in visual acuity, and that the color sense is not impaired.

Some authors, particularly Nothnagel, located in the pons a so-called *spasm centre*, stimulation of which excites general convulsions of an epileptic type. Later this view has been discarded, without it being denied that general motor symptoms of irritation may be excited in the pons. Bechterew, who confirmed Nothnagel's observations, showed that the spasms appearing in a lesion of a definite pontine area (vasomotor centres ?) are derived from the cerebrum.

This description suffices to show how varied is the symptomatology of pontine diseases, but still in most cases so characteristic that the symptoms are a certain guide-post to the location of the disease.

The pons is a *favorite seat of foci of softening*, while hemorrhages are not so frequent. Glioma and tubercle are found in this region. *Encephalitis*, particularly the forms resulting from infectious diseases (influenza ?), often affects the pons. Multiple sclerosis almost always involves the pons.

It should be remembered that an aneurism of the basilar artery may compress the pons and cause pontine symptoms.

The following case will serve as an example of pontine disease:

Thirteen-year-old F. complained, several weeks after an attack of influenza, of formication and heaviness in the left half of the face. Several days later weakness of the right arm and leg manifested itself. Then came an impairment of hearing, ataxic gait, and diplopia. The speech became indistinct.

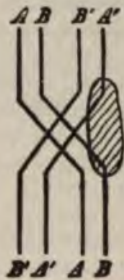
In the examination undertaken several weeks after the beginning of the disease I found a *paralysis of the left facial* in all its branches, with partial reaction of degeneration, hypesthesia of the *left trigeminal area*, a paralysis of the *left abducens*, with inability to rotate both eyes towards the left. The hearing was lessened on the left side; osseous conduction was somewhat impaired. On the *right* half of the body there was a *paresis*, with rigidity and increased knee-reflexes. Sensation was diminished on the right arm and leg, less so on the left arm. In addition, *ataxia* was present in the right arm, less so in the leg, and a trace in the left arm. Speech was somewhat *nasal* and indistinct, and *deglutition* was somewhat hindered. No alterations of the fundus. Slight headache, no vomiting.

These phenomena were caused by an encephalitic lesion which embraced most of the left half of the pons and extended from the acoustic nucleus to that of the abducens. As it overstepped the median line below, it involved the right lemniscus. The pyramids were affected only for a short distance.

In the *medulla oblongata* the different tracts and centres are embraced in a very small space, so that lesions of relatively slight extent may produce important disturbances.

The motor conducting tracts lie close together here, being called the pyramidal tracts. If they are injured above the decussation, hemiplegia of the contralateral side occurs; paraplegia of all four extremities in a bilateral break of conduction. A lesion which is located at the pyramidal decussation may involve the fibres of one side before, those of the other side after the decussation, and thereby produce the rare phenomenon of *hemiplegia cruciata* (Fig. 249). The pyramids at all heights probably send fibres to the motor nerve nuclei of the medulla oblongata, which cross the median line in the raphe. The *sensory* tracts are found partly in the lemniscus or the olivary midlayer, partly in the reticular field and in the restiform body. We have already spoken of the uncertainty which exists in our knowledge of this field. Clinical observations (Senator, Goldscheider, Reinhold, v. Oordt, and others) and experimental observations (Bogatschow) render it probable that the tract for the muscular sense decussates in the medulla oblongata and reaches with the internal arcuate fibres the fillet of the other side, and in the midolivary layer runs close to the raphe, and that, therefore, the posterior column lemniscus tract contains these fibres. A lesion of it would, therefore, cause hemiataxia of the opposite side, while foci of disease in the lower section of the oblongata may entail ataxia of the same side. Homolateral ataxia is probably also evoked by lesions of the restiform body and direct cerebellar tract. There is no doubt that a lesion of this part can produce cerebellar ataxia (perhaps, also, nystagmus and vertigo).

FIG. 249.



Schematic representation of hemiplegia cruciata (crossed hemiplegia). *A*, right-sided brachial path; *B*, right-sided crural path; *A' B'*, corresponding left-sided paths. Shaded portion shows the injured left-sided brachial and the right-sided crural paths.

The tracts for the senses of pain and temperature probably decussate in the lower part of the medulla oblongata and in the reticular field (ventral part), or are also contained in the fillet.

Whether the fibres for tactile sensation are also found here or in the midolivary layer is not certain; they appear, however, to run mesad to those for the pain and temperature senses. This is all mere hypothesis, however. Observations concerning the function of the olive and the symptoms produced by a lesion of it are especially lacking. A centre for deglutition has been located in the olive (and accessory olive) (Schroeder van der Kolk, Kesteven, Moeser), though this has not been definitely proven. Bechterew regards the olive as a co-ordinating centre, though injury to it does not always cause incoördination.

It is noteworthy that some of the *nerve nuclei* in the oblongata lie *closely alongside of each other*, so that a lesion the size of a pea may cause a bilateral paralysis of these nerves. This is particularly true of the *nuclei of the twelfth cranial nerves.*

Of the trigeminal we find here only the spinal sensory root, which lies so close to the major sensory conducting tract that a relatively small lesion may produce a *hemianesthesia cruciata* or *alternans,—i.e.*, an anesthesia in the trigeminal on that side and of the body of the opposite side. In a case observed by me, a small lesion of the oblongata produced *hemiparesthesia cruciata* with pain in the same territory.

There is also a *bulbar ataxia*, though it is not certain what part must be injured to evoke this symptom.

Focal diseases of the medulla oblongata often produce alternating paralysis. Unilateral lesions may paralyze the eighth to the twelfth cranial nerves, while hemiplegia occurs on the opposite side; hemianesthesia alternans may be combined with it.

The medulla oblongata also contains a number of *centres* which act partly reflexly, partly automatically. These are in part identical with the nuclei which lie upon the floor of the fourth ventricle, though their relations are by no means clearly understood. Behind the place of exit of the vagus on both sides of the posterior apex of the fourth ventricle is the place Flourens determined upon as the location of the respiratory centre. Others regard the *solitary bundle* as having a connection with this centre. Misslawski described a nucleus in the inner part of the reticular field mesad to the roots of the twelfth, which is supposed to have some influence upon respiration.

Respiratory centres are also said to have been detected in the thalamus, upon the floor of the third ventricle, and in the posterior quadrigemina (postgemina).

The *cardiac inhibitory centre* lies near the respiratory centre. Under the floor of the fourth ventricle lies the centre for *deglutition*, which is stimulated through the sensory pharyngeal and palatal nerves. Perhaps the nucleus ambiguus may form this centre. Even a unilateral lesion of it is said to cause deglutitory disturbances (v. Oordt, Schlesinger). Only a part of the act of deglutition, that which occurs in the mouth, is under volitional control. As soon as a morsel enters the pharyngeal space, deglutitory movements become reflex.

The musculature of the lips, tongue, palate, and larynx governing *articulation* and *phonation* are innervated from nuclei in the medulla oblongata and pons, which are at the same time trophic centres for these muscles.

A centre for *vomiting* is also found in the medulla oblongata near the respiratory centre. The seat of the centre for salivary secretion is not known.

Injury of the lower part of the fourth ventricle can, as is well known, cause *diabetes mellitus* (Claude Bernard). It is probably the vasomotor centre which is affected. Pathology teaches us that diseases of the medulla oblongata occasionally evoke *mellituria*. It has not yet been investigated thoroughly, however; and we do not know just how it is caused. *Polyuria* and *albuminuria* have been noticed in a few cases of diseases of this part of the brain.

We know as little concerning the location of the *vasomotor centres* in the medulla oblongata. The *lower central nuclei* have been referred to as such by Bechterew, but others have indicated other parts. Reinhold has recently studied this question, and considers the median areas of the subependymal ventricular gray of a relatively large extent as vasomotor centres.

A number of diseases of the central nervous system involve the medulla oblongata without it being the chief centre of the disease; among these are tabes dorsalis, gliosis, and multiple sclerosis. A myelitis of the upper cervical region may also extend to the medulla oblongata. *Progressive muscular atrophy* may involve systematically the medulla oblongata; it almost always becomes affected in *amyotrophic lateral sclerosis.*

These diseases are discussed at other places. We will notice here only those diseases which commence in the medulla oblongata or extend to it from the immediate vicinity.

PROGRESSIVE BULBAR PARALYSIS (PROGRESSIVE GLOSSOPHARYNGOLABIAL PARALYSIS).

This is a rare disease. It attacks those of from fifty to sixty years of age. It has appeared in few people before the fortieth year of life. Its occurrence in children has been noted in a few cases which have not been cleared up by any post-mortem observations. This infantile form, however, deserves a special description.

The causes are unknown. Exposure to cold, emotional shock, traumata, and particularly over-exertion of the muscles of the tongue, lips, and palate have been mentioned. In one case (Hoffmann) chronic lead intoxication was present. Whether heredity or a congenital predisposition is in play is doubtful.

Symptomatology.—A *slowly* developing *speech disturbance* and *impairment of deglutition, mastication, and phonation,* due to a *progressive symmetrical paralysis* and *atrophy of the labial, glossal, palatal, pharyngeal, laryngeal, and masticatory musculature* form the basis of this disease.

The speech disorder is, as a rule, the earliest symptom. The patient notices that speaking, particularly a long conversation, tires him, and that the words are not enunciated as distinctly as heretofore. Others also observe these changes. The linguodentals and linguopalatals (d, t, l, r, u, s, ch, i) are the earliest involved. Later on the labials are also lost (p, w, f, m, o, and u), particularly those which demand tight closure of the lips. At the same time or later a *nasal tone* occurs, in that a part of the expiratory air passes through the nose, *b* and *p* sounding like *mb*, *mp*. The bulbar speech disturbance, *dysarthria*, can now no longer be mistaken, the words are pronounced indistinctly, run together, and a more or less marked *nasal tone* appears. The patient speaks as if he had a dumpling in his mouth. It increases during a conversation, as exhaustion increases the disturbance.

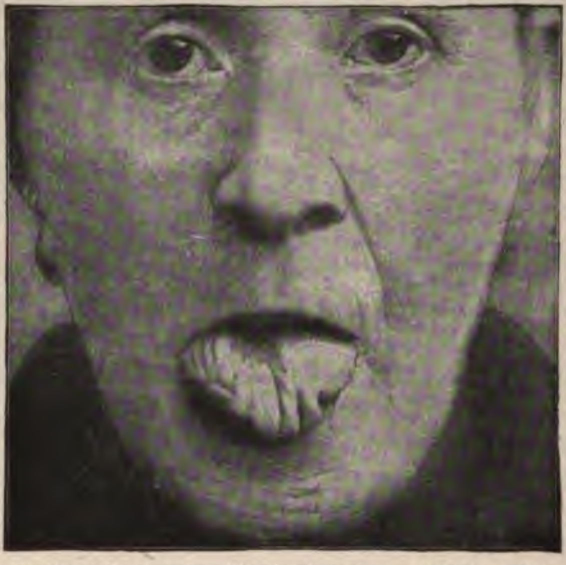
In the first stage, or after the dysarthria has been present some months, *deglutition* becomes impaired. The patient cannot easily bring the bolus into the pharynx and from here into the esophagus, fluids regurgitate through the nose or reach the laryngeal opening and produce attacks of vomiting, and finally the dysphagia is converted into complete aphagia or inability to swallow solids or fluids.

Mastication is also not rarely involved in the further course of the disease.

Phonation and *respiration* finally suffer. The voice becomes weak and monotonous, cannot be modulated, is deep, and hoarseness may come on and increase to aphonia. The fits of coughing are weak and silent. Respiration generally becomes difficult and dyspneic near the end of life, and choking spells are not uncommon.

The disturbances described are due to *muscular paralysis* and *degeneration*. The tongue is first affected in most cases. The coarser movements need not be impaired at first, even though glossal articulation is distinctly hindered. The weakness by and by becomes apparent upon examinations of the motility. The tongue can be protruded but partially and slowly, is instantly retracted again, and cannot be properly moved sideways. In the mouth it lies on the floor, a flabby, perhaps atrophic, rugose mass. The weakness of the labial muscles is shown by their weak closure and by inability to whistle and to point the lips. The velum hangs deeply and is not lifted well during phonation or, later, not at all. The pharyngeal reflex is, as a rule, abolished. The paralysis extends later on to the muscles of deglutition.

FIG. 250.



Atrophy of the tongue and lips in glossopharyngolabial paralysis.

A laryngoscopic examination generally reveals at first a normal picture, but later paresis of the adductors may be recognized by the half-open glottis during phonation. Weakness of the masticatory muscles is revealed by the inability to move the lower jaw lateralward and by the weak closing of the jaw.

Glossopharyngolabial paralysis is a degenerative paralysis. The atrophy does not, as a rule, come on early, and does not keep pace with the paralysis. The disturbance of function is prone to precede a noticeable muscular emaciation a long time.

It is first detected on the tongue; this becomes flabby, feels soft and spongy, and the fibrillary tremor is very noticeable. If the atrophy progresses, furrows and depressions appear (Fig. 250); the tongue finally becomes smaller *in toto*, though with normal volume it may already be histologically altered. The atrophy spreads later to the lips, but is not so pronounced here; the lips become thin and no longer feel muscular. Atrophy of the masticatory muscles is rarely pronounced, though a fibrillary tremor is often early noticed in these muscles.

The muscular degeneration produces alterations of the electric excitability, generally a *partial reaction of degeneration*, which may be hidden for some time by the normal reaction of the still intact muscular fibres, and which is generally only detected in the later stages and must be carefully searched for. In an advanced stage the *facial expression* reveals the disease. The mouth is open, the lower lip droops, the oral angle is drawn downward, and saliva, secreted either in normal or in exaggerated quantities, drools from the mouth. The lower half of the face is fixed and expressionless; the upper half, from the normal motility of the eyes and the upper facial muscles, alone betraying the sound mind and normal intellect. In a few cases, however, as in one observed by myself (and Remak), the upper facial region also becomes affected.

The patient cries easily, the mouth not being distorted much thereby, while the respiratory muscular action assumes a spasm-like character, and is occasionally combined with an inspiratory stridor. The tongue now lies inert on the floor of the mouth, speech becomes unintelligible, as articulation is almost entirely abolished; swallowing is impossible; breathing accelerated; the pulse often increased to from one hundred and twenty to one hundred and forty beats a minute; and the patient is noticeably emaciated and is in a half-helpless condition.

The symptoms described depend upon a paralysis of the motor cranial nerves. This palsy in typical cases, however, does not involve the nerves for the ocular muscles; and we wish particularly to emphasize the fact that the sensory cranial nerves and those for the special senses are never affected.

In some cases, however, a *spastic* condition may be observed, in that the deep reflexes of the facial and jaw muscles are exaggerated (page 164).

Just as bulbar paralysis may be secondary to progressive muscular atrophy and amyotrophic lateral sclerosis, so can these diseases join themselves to an existing bulbar paralysis without reaching a developed stage. It is, for instance, not uncommon to find increased deep reflexes of the extremities and an atrophy of some muscles of the hand and arm in addition to the phenomena of glossopharyngolabial paralysis.

Progressive bulbar paralysis has an insidious onset and a slowly developing course. At times deceitful periods of standstill, rarely remissions, occur.

Only in a few cases has an acute (though not apoplectic) onset of the speech disturbance been noticed; the further course was, however, a steadily progressive one. The disease, so far as we know, always ends in death, generally after one to three years, sometimes later. Many cases are known in which death resulted within the first year.

Inanition, aspiration pneumonia, asphyxia, or any intercurrent disease may be the cause of death.

Diagnostic Remarks.—Before a diagnosis of bulbar paralysis is made by reason of a bulbar speech, satisfy yourself that the speech defect is not due to a mechanical hinderance (defective palate, etc.). A paralysis of the palatine velum does not justify a diagnosis of this disease, as it may occur in other affections,—for instance, in diphtheria. When, however, it can be determined that the speech defect has developed insidiously and has gradually increased, and when paralytic symptoms have appeared in different muscle regions of the articulatory apparatus, the diagnosis of progresive bulbar paralysis can be made contingently and, if atrophy comes on, with certainty. As long as atrophy is absent, confusion with benign forms (see Bulbar Paralysis without Anatomic Lesions) is possible. Special caution is necessary in youthful persons, as there are certain curable forms of polio-encephalitis whose course resembles that of atrophic bulbar paralysis. Even if here also the symptomatology is generally different and the development acute or subacute, an insidious course sometimes occurs.

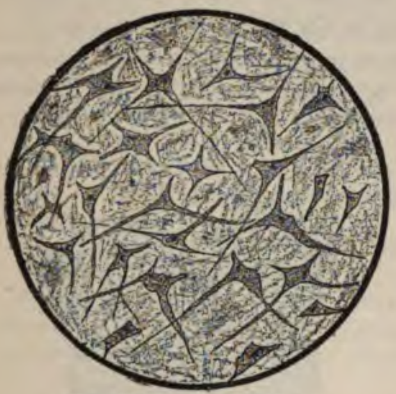
Differentiation from acute bulbar paralysis and pseudobulbar paralysis is generally easily made, though some cases—to be discussed below—offer many difficulties.

Tumors of the medulla oblongata produce symptoms of bulbar paralysis of gradual development, but the paralytic symptoms are not spread symmetrically, the elective character—involvement of only the motor nuclei—is not present, and signs of intracranial pressure, which are absent in cases of true bulbar paralysis, are found.

Pathological Anatomy.—The basis of this affection is a disease of the motor nerve nuclei which are found in the medulla oblongata and pons—the hypoglossal and facial nuclei, the motor glossopharyngo-vago-accessorius, and occasionally the motor trigeminal. The ganglion cells, which are the most important components of these nuclei, gradually degenerate, lose their processes, and finally disappear entirely.

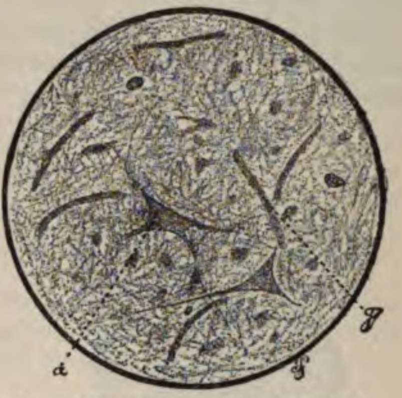
(Figs. 251*a* and *b*, 252*a* and *b*.) The same is true of the intranuclear net-work and of the intra- and extra-bulbar roots. Atrophy of the latter is sometimes recognizable macroscopically. It is evident that the degeneration involves the muscles and intramuscular nerve branches.

FIG. 251*a*.



Normal hypoglossal nucleus.

FIG. 251*b*.

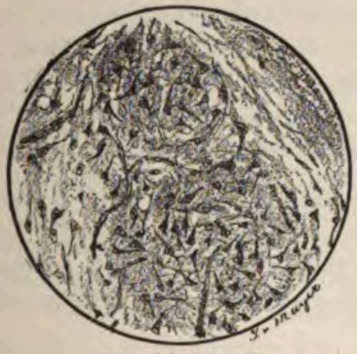


Atrophic hypoglossal nucleus in bulbar paralysis. *g*, blood-vessel.

(Carmine preparations.)

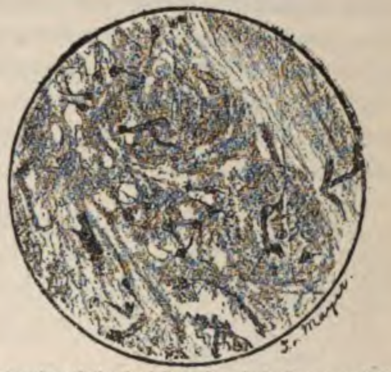
The disease may confine itself to the gray matter named above, as in some of the cases of Charcot, Duchenne, Joffroy, Duval, Raymond, etc. The pyramidal tracts are, however, often involved (Fig. 253*b*);

FIG. 252*a*.



Normal facial nucleus.

FIG. 252*b*.



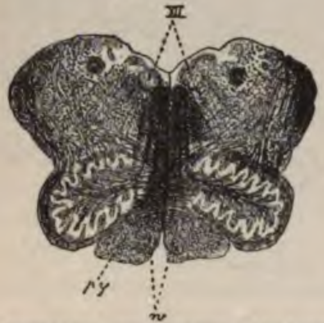
Atrophy of the facial nucleus in bulbar paralysis.

(Carmine preparations. Enlarged less than Fig. 251.)

the sclerosis of the motor conducting tract appears in some cases to precede the nuclear atrophy; it may, however, be entirely absent, and the course is said to be more rapid in those cases in which only the gray matter is affected. The characteristic alterations of progressive muscular atrophy and of amyotrophic lateral sclerosis are often found in the spinal cord.

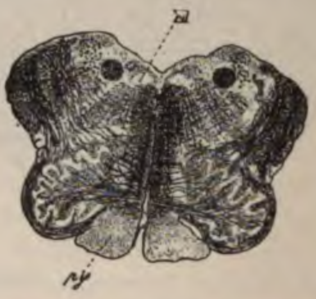
Treatment.—The disease, as far as we know, is incurable. We do not even have any definite ideas concerning prophylactic measures. Silver nitrate, strychnine, iodide of potassium, and arsenic have been recommended. Atropine in doses of one-half of a milligramme ($\frac{3}{400}$ of a grain) has been given with good results for the salivary drooling. A strengthening nourishment must be seen to early. Artificial feeding through a sound may be necessary. Electricity should be used in every case. The medulla oblongata should be directly galvanized, the current being transmitted from one mastoid process to the other, a rheostat being used. (Strength of current about two or three milliamperes, with an electrode of fifty square centimetres.) Galvanic and faradic stimulation of the labial, glossal, and palatal muscles. Production of the *galvanic deglutitory reflex* by stimulation of the neck, the anode on the nucha, the cathode rubbed over the sides of the neck.

FIG. 253*a*.



Normal medulla oblongata at the height of the hypoglossus. xii, hypoglossal nucleus; *w*, hypoglossal roots; *py*, pyramids.

FIG. 253*b*.



Atrophy of the twelfth nucleus and its roots, also of the pyramids, in amyotrophic lateral sclerosis.

(Weigert's stain.)

INFANTILE (HEREDITARY, FAMILIAL) FORM OF PROGRESSIVE BULBAR PARALYSIS.—Fazio, Charcot, and Londe, particularly, have described a form of progressive bulbar paralysis of childhood. Berger, J. Hoffmann, and Remak have described cases which cannot be definitely included here.

The disease was characterized particularly by its hereditary, familial type, it having occurred in children of parents who were blood-relations. Stigmata of heredity and degeneration (prognathism, etc.) have also been found. Another peculiarity is that the paralysis commenced in the *upper facial area*, and involved this region especially (lagophthalmos, etc.). Ptosis also occurred.

The paralysis was an atrophic one with decreased electric excitability and a partial reaction of degeneration. Articulation and deglutition were involved in a characteristic manner. Paresis of the laryngeal muscles was present (adductors), while the extremities were not involved. The *anatomic* basis is unknown, though nuclear disease (*poliencéphalite moyenne*) has been presumed. Notwithstanding its similarity to certain forms of myopathy, a primary muscular affection can hardly be in question. (See page 171.) On the contrary, it cannot be denied that the disease is similar to the form of congenital facial diplegia and oculomuscular paralysis due to "infantile nuclear atrophy" (pages 281 and 292), as the condition here, though due to congenital developmental anomalies, only manifests itself later in childhood, while in the other it is congenital. Möbius considers the cases in which the disturbances described develop in childhood as being cases of the so-called infantile nuclear atrophy.

A congenital paralysis of the bulbar nerves has been described by Berger and Hoppe-Seyler. I will speak below of another form of infantile glossopharyngolabial paralysis. A hereditary disease with symptoms of atrophic bulbar paralysis has been observed in adults by Bernhardt.

Bulbar symptoms, of unknown genesis, were observed by me accompanying a laryngismus stridulus.

The so-called *unilateral bulbar paralysis*—cases have been described by Pel, Erb, Wiener, Geronzi, and others—approaches, generally, the acute form, and must be differentiated from Duchenne's disease. (Compare the next chapter.)

ACUTE APOPLECTIC BULBAR PARALYSIS.

The symptom-complex of glossopharyngolabial paralysis may develop acutely. The pathologico-anatomic alterations which cause this acute form are of a varied nature. It is, however, generally an affection arising in the vascular apparatus—hemorrhage, or, more often, *softening* after *thrombosis;* more rarely, emboli of the *vertebral* and *basilar* arteries and their branches. This form of bulbar paralysis has been especially studied by Lichtheim, Leyden, Senator, and myself.

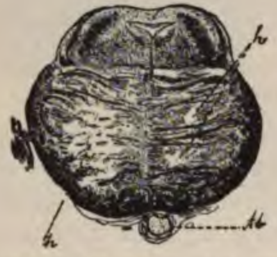
Hemorrhages which enter the substance of the pons and of the medulla oblongata are, on the whole, rare, and generally evoke death quickly, before the signs of bulbar paralysis have become fully developed. Isolated and multiple hemorrhagic foci in the pons and in the medulla oblongata have been observed in some cases (Senator, Schulz, Schlesinger).[1]

The causes are those of cerebral hemorrhage, though it must be particularly emphasized that cranial injuries also, especially those affecting the occipital region, may cause hemorrhages of the pons and of the medulla oblongata. Foci of softening occur more often in these regions. They may be of microscopic size or so large as to extend from the upper region of the pons down into the medulla oblongata, and also take up a large part of the cross-section. Small foci are often found in addition to larger ones. Softening is almost always a result of the occlusion of one of the vessels nourishing the bulb. The basilar, also the vertebral (especially the left), are thus often closed by a thrombus. The thrombosis is the result of an arteritis, either of atheroma or of a specific vascular disease, which involves particularly the basilar (Fig. 254) and the vertebral arteries. Emboli more rarely reach the vertebral (generally the left) from the diseased heart. In other cases, not these arteries themselves but their branches become occluded. It should be remembered that arteriosclerosis does not only injure the afterbrain by thrombosis of the vessels, but the circulation itself is impaired by the narrowing of these vessels and the disease of their walls. The atheromatous artery is so often dilated and also so rigid that it exercises pressure upon the neighboring parts of the afterbrain (Fig. 257). Only in a few cases were foci of softening found in the pons or in the oblongata without any corresponding vascular disease; they were referred to an encephalitis or bulbar myelitis (Leyden, Etter, Eisenlohr, myself). A hemorrhagic encephalitis of this nature has deen described by Schlesinger and Hori.

---

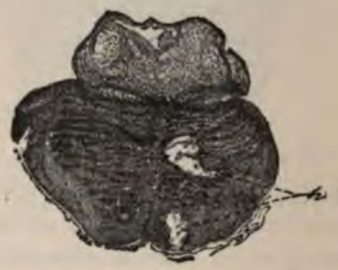
[1] In gliosis, also, acute bulbar paralysis may come on suddenly from hemorrhage. Schlesinger has also observed similar symptoms in a few cases of caisson paralysis.

FIG. 254.



Thrombosis of the basilar artery, with foci of softening in the pons (syphilitic infection). *h*, lesion. (Pal's stain.)

FIG. 255.



Two foci of softening (*h*) in the pons after vascular disease.

An *acute polio-encephalitis inferior* (and subacute)—*i.e.*, an acute and subacute inflammation of the gray matter of the bulbar nuclei with the symptom-complex of bulbar paralysis—also occurs. (Compare chapter on encephalitis.) The process has been brought into analogy with acute poliomyelits. It has also been sometimes observed in childhood and following upon infectious diseases (Eisenlohr, Hoppe-Seyler, Geronzi). The affection may also confine itself to one side and evoke a unilateral bulbar paralysis.

A few recent observations have confirmed the view that the bulbar symptom-complex has been due, in a few cases, to an *abscess* of the medulla oblongata (Eisenlohr, Schlesinger, Lorenz). Bulbar neuritis and bulbar neurosis will be discussed later.

In describing the symptoms we intend to confine ourselves to typical cases,—*i.e.*, to those caused by softening (and hemorrhages). *Prodromes* occur occasionally, symptoms directly produced by the vascular disease. They include headache, particularly occipital and nuchal pain, vertigo, sleeplessness, tinnitus, and a flickering before the eyes.

The *paralysis* manifests itself *suddenly*. A vertiginous attack, more rarely an apoplectic stroke with complete unconsciousness, opens the scene. The vertigo may be so severe that the patient falls to the ground. Vomiting may accompany it. In a few cases general convulsions of an epileptic character came on. Soon after this stroke the fully developed symptoms of a glossopharyngolabial paralysis appear. Only rarely do we find a few days pass before the acme of the disease is reached; and a subacute course—extending over weeks—has been observed in very few cases of encephalitis or of myelitis and bulbar polioencephalitis. *Dysarthria*, or even anarthria, *dysphagia*, or total inability to swallow, are initial symptoms. Closure of the jaws, due to a tonic contraction of the masticatory muscles, is one of the earliest phenomena. In the further course of the disease, sometimes from the beginning, a weakness of the masticatory muscles is noticed. The musculature of the lower facial area is more or less completely paralyzed on both sides, though the paralysis is generally asymmetrical.[1] Glossoplegia and paresis of the palatal and pharyngeal musculature come on. The laryngeal muscles are occasionally affected on one or both sides; this is particularly true for the adductors, paralysis of which causes hoarseness or aphonia. The acoustic is only rarely involved.

In most cases the paralysis extends to the muscles of the extremities. A paraplegia of the arms and legs or a hemiplegia occurs from the beginning, generally upon the side upon which the bulbar nerve paralysis is the least pronounced—a kind of alternate hemiplegia; or the opposite occurs, or we find a paralysis of one arm and both legs. Paraplegia of the lower limbs with slight paresis of the upper is not uncommon; the opposite has been seen only once by me. The hemiplegia may also jump from one side to the other. Paralysis of the extremities is combined with rigidity of the muscles and increased tendon-reflexes. *Respiratory disturbances* (dyspnea, Cheyne-Stokes breathing, etc.) may be present from the beginning or develop later; they generally only manifest themselves near the end of life. An *acceleration of the pulse* and an *increased body temperature* to 102° or 104° F. are not uncommon; a marked rise is, as a rule, observed only in lethal cases, near the time of death.

---

[1] Motor symptoms of irritation in the facial area are rarely caused by these acute affections of the pons, though I once observed a marked convulsive tic in addition to the characteristic paralytic symptoms in a pontine lesion of embolic or encephalitic origin.

The *facial expression* of the patient is altered in a characteristic manner (Fig. 256); facial immobility is the more pronounced because the disease comes on so quickly. Though the sensorium generally remains free and the intellect is not impaired, one is easily inclined to regard the patient as mentally weak, because the slightest cause brings on a spell of *crying* (more rarely, of *laughter*). This has a spasm-like character, and produces a tonic contraction of the mimic and respiratory muscles, to which a condition of dyspnea may be added.

It is comprehensible that paralyses of the labial, glossal, and palatal muscles are at first not combined with atrophy, as this requires time to develop; an atrophy, however, rarely manifests itself at any time in the course of the disease, and then only in one nerve area (half of the tongue or the facial muscles of one side).

This is due to the fact that the focal lesion generally does not injure the nucleus and the roots coming from it, but the tract passing from the brain to the nucleus—the corticonuclear or supranuclear conducting tracts—on its way through the pons and shortly before its entrance into the gray matter of the nucleus.[1]

Naturally a part of the nuclear area may also become involved, but rarely to such an extent as to entail trophic disturbances. Atrophy then occurs accompanied by diminished reflexes in the affected areas. I found, for instance, in such a case atrophy of one half of the tongue; Reinhold, loss of electric excitability on one side of the pharynx. The few cases in which we have an acute or subacute polio-encephalitis, an inflammation affecting only the gray nuclear substance,—*e.g.*, the bulbar form of infantile palsy,—are exceptions. In a case of this kind there was a total facial paralysis with reaction of degeneration; in others, there was lingual hemiatrophy which (*e.g.*, in the cases of so-called unilateral bulbar paralysis) formed the most constant symptom, or several cranial nerves of one side were affected. Wiener and Geronzi have found corresponding anatomic lesions.

These cases of acute and subacute polio-encephalitis are rare, and can be differentiated from acute bulbar paralysis by their non-apoplectic development. (See also page 531.)

Sensory disorders may belong to the phenomena of acute bulbar paralysis; patients not rarely complain of paresthesia on one side of the body or in one or both arms, and at times of severe *pains* on one side of the body. Hypesthesia or *anesthesia* in a trigeminal area (several times, with the exception of the *mucous membranes*) and *hemianesthesia alternans* or *cruciata* (Senator) have been observed. Bulbar ataxia has already been mentioned. Salivary dribbling occurs often; albuminuria, mellituria, etc., rarely.

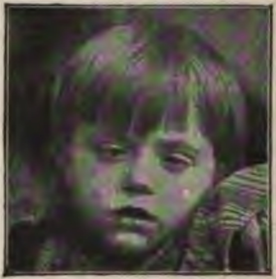
---

[1] These supranuclear forms should really be included under pseudobulbar paralyses, as, for example, Schlesinger does, though I regard it as better to confine this name to cerebral glossopharyngolabial paralyses (due to lesions in the cerebral hemispheres and central ganglia), as it is not possible to make this distinction in forms due to foci of softening in the pons and medulla.

If the lesion extends to the upper part of the pons or to the quadrigeminal region, *oculomotor* and other symptoms manifest themselves.

It can be seen that the symptomatology must be a variable one, according to the size, seat, and extent of the lesion. Cases have been described in which only the vagus, accessorius, and trigeminus of one side were affected; others, in which, at the same time, the sensory and motor conducting tracts were involved; others, again, in which most of the corticonuclear fibres and the pyramidal tracts of both sides were divided, and in addition the facial nucleus injured entirely or partially. In a case observed by Elsholz, notwithstanding an extensive pontine hemorrhage, only cranial nerves were paralyzed, while the extremities were not affected. If the necrobiosis confines itself to one side, it produces the different forms of hemiplegia alternans. In cases in which the *restiform body* was affected, disturbances of co-ordination, especially swaying and falling towards one side, were noticed several times; a *rotatory nystagmus* has also been supposed to be due to this affection. It appears as if the acute focal diseases of the pons may also induce a transient loss of the knee-reflex (?). A focus in the pyramidal tract may involve the brachial fibres before decussation, the crural fibres after decussation, and so cause symptoms of *hemiplegia cruciata*. It should be remembered that the foci are generally of an irregular shape, their chief extension being in the dorsal area of one side of the pons, only extending slightly into the basal region of the other side. All this clearly shows why the paralytic symptoms are rarely developed as symmetrically as in Duchenne's bulbar paralysis.

FIG. 256.



Facial expression in paralysis of both facial nerves, abducens nerve, and the masticatory muscles from a diffuse pontine disease.

Symptoms of acute bulbar paralysis appear in full intensity and in complete development in those cases in which the vertebral or basilar artery is closed by a thrombus or embolus; while the occlusion of small branches evokes only circumscribed paralytic phenomena.

Notwithstanding the careful studies of Duret of the vascular supply of the afterbrain, and the efforts of later authors (*e.g.*, Wallenberg), a definite symptom-complex evoked by occlusion of the different arteries cannot be established, as the course, relative calibre of the arteries, and the vascular supply of different areas vary so pronouncedly. The nuclei and their roots are, moreover, not always supplied by the same arteries. As a rule, the vertebral supplies the nuclei of the hypoglossus and accessorius through the anterior spinal artery. Vessels arise from the upper part of the vertebral or from the lower part of the basilar for the nuclei of the vagus, glossopharyngeal, and acoustic, though it appears that the ambiguus nucleus is supplied by the inferior cerebellar artery (*i.e.*, from the vertebral artery). The abducens nucleus receives its branches from the basilar artery. Occlusion of one *vertebral* will, therefore, impair the function of the nuclei of the ninth to the twelfth cranial nerves and the spinal fifth on one side. When there is only one anterior spinal artery arising from only one vertebral, both sides are more or less involved. The extremities may also be variously affected, as the pyramidal tract is supplied by the anterior spinal artery (more rarely by the vertebral), generally from the corresponding branches in question, before anastomosis with the opposite side. Obliteration of the vertebral artery will cause hemiplegia of the contralateral side when the part lying above the decussation is softened, on the same side when the softening affects the distal part. If only one spinal artery is present, and if this comes from only one vertebral artery (it is then generally the left), obliteration of it may cause a bilateral hemiplegia. Bilateral hemiplegia would also occur if the softening were a unilateral one but involving the corresponding pyramidal tracts above and below the decussation. If the pyramidal tract is supplied by the united anterior spinal artery, occlusion of the vertebral need not produce any paralysis of the extremities. If the closure is in the upper part of the basilar artery, the ocular muscles, at least the abducens (and upper facial?), according to Duchenne, are affected. Obliteration of the basilar artery denotes, therefore, a more severe affection, as it is a single artery and its branches supply the respiratory centres and tracts. Duret claims, therefore, that thrombosis of its lower part is immediately fatal.

The *inferior cerebellar* artery also seems to be often affected by emboli or thrombi; at least the clinical observations of Wallenberg, v. Oordt, and others allow us to presuppose this. The part affected lies in the lateral sections of the medulla oblongata, and includes the restiform body, the motor vago-glossopharyngeal nucleus (?), the spinal trigeminal roots, and a part of the reticular field, including the internal arcuate fibres. The olive may eventually become affected also.

The clinical picture includes *unilateral palato-laryngeal paralysis*, *dysphagia*, perhaps *anesthesia* in the region innervated by the fifth nerve on the same side and hemianesthesia on the opposite side of the body (hemiplegia only, when the pyramidal tracts are affected). Several times an ataxia on movement was found in the arm of the same side (Proust, Dumenil, Reinhold), which was thought to be due to the involvement of the restiform body, or of the direct cerebellar tract, or of the internal arcuate fibres before their decussation. Vertigo, falling to one side, and nystagmus seem to occur also. Wallenberg has studied this question most carefully, and has sought to bring the symptoms into accord with the circulatory relations.

Foci of softening, which develop in circumscribed loci in the medulla oblongata, may produce unilateral hypoglossal paralysis (with atrophy), and cause crossed paralysis, as was determined by myself, Reinhold, and particularly by Révilliod-Goukowsky. An exact topical diagnosis cannot always be made in lesions of the pons and medulla, though we can say that lesions of the pons produce symptoms which are not observed in affections of the medulla oblongata (paralysis of the ocular muscles, myosis, total degenerative facial paralysis, associated paralysis of the eyes).

The course is dependent somewhat upon the severity of the phenomena. Basilar thrombosis is almost always fatal. It is possible, however, that syphilitic endarteritis of this blood-vessel may cause only a transient total obliteration, circulation being resumed before necrobiosis develops. Excluding the most severe cases, in which death results in a few days or weeks from aspiration pneumonia, respiratory or cardiac paralysis, we can characterize the course of acute bulbar paralysis as *retrogressive;* the phenomena reach their height at an early stage, while improvement comes on in their further course, which may progress to recovery. That such a course is possible in the most severe cases is shown by a case described by myself; in it an until then healthy man suddenly developed the signs of *glossopharyngolabial paralysis* with *paraplegia of all four extremities.* There were complete *anarthria* and *dysphagia, tonic contraction of the lower jaw, accelerated pulse, increased temperature, profuse salivation* (particularly upon electric stimulation of the facial muscles), etc. The condition was so much improved in from five to six months that a left-sided hemiparesis and a slight nasal tone were the only residual symptoms of the disease, and to-day (fourteen years after) the patient still enjoys good health outside of these slight disorders.

The treatment of acute bulbar paralysis is, in general, similar to that of hemorrhage, softening, etc.

An *antisyphilitic* treatment is generally justifiable or demanded. Acute encephalitis of the pons and medulla is to be treated, like encephalitis in general, by antiphlogosis and purgation. In one case enormous doses of calomel were administered with an excellent result. Much stress must be laid upon *nutrition.* Deglutitory paralysis makes the use of a sound necessary. The danger of aspiration pneumonia must be prevented. It appears that these patients are less resistant to croupous pneumonia. In the later stages electric stimulation in the manner described for the chronic form is advisable.

Eisenlohr has lately described a peculiar, essentially bulbar symptom-complex which occurred in youthful individuals in the course of *typhoid.* There were present dysarthria, due to paralysis of the lips, tongue, and palatal muscles, dysphagia, paresis of the masticatory muscles, weakness in all the extremities, stupor, and in one case optic neuritis. Two of those affected recovered. In the lethal cases *streptococci* were found in different parts of the central nervous system—a form similar to but not identical with streptococcus citreus—without any pronounced histologic alterations. Eisenlohr regards the phenomena as being due to a *mixed infection.*

Seitz has also found in a case of bulbar paralysis bacteria (pneumococci) throughout the brain.

ACUTE BULBAR NEURITIS.—*Multiple neuritis* may also affect the nerves coming from the medulla oblongata. Symptoms of glosso-pharyngolabial paralysis then occur in conjunction with multiple neuritis (Kast, Eisenlohr).

Eisenlohr has also observed a severe form of bulbar paralysis occurring in the course of a case of *leukemia*. Dysarthria, dysphagia, bilateral total facial paralysis (reaction of degeneration), anesthesia in the region of the fifth cranial nerve, ageusia, etc., were present. After four weeks death came on with dyspnea and collapse; and multiple hemorrhages were found in the sheaths of the bulbar nerves, also a profuse dense infiltration of them with lymphoid elements. A similar case was described by W. Müller; degenerative processes in the medulla oblongata have also been observed in leukemia.

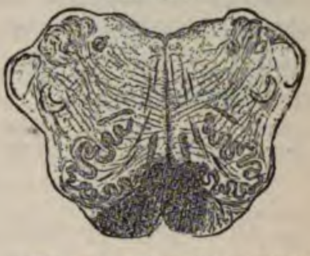
COMPRESSION BULBAR PARALYSIS.

Tumors which extend to the medulla oblongata or its vicinity (basis, cerebellum) may evoke in an acute manner the symptoms of bulbar paralysis; the development of the whole disease is, however, generally a protracted one; and the bulbar symptom-complex also does not come on suddenly, but *step-like;* or the until then slight paralytic symptoms suddenly exacerbate. An acute onset of bulbar paralysis from compression was observed by me in a case of gummatous meningitis of the posterior cranial fossa. Caries of the upper cervical vertebræ and of the occipital bone (Vulpius) may also cause this symptom-complex.

Aneurisms of the basilar and vertebral arteries, whose symptomatology is principally due to the compression exercised upon the pons and oblongata, and the nerves arising from them, are particularly interesting in this connection (Lebert, Griesinger, Gerhardt, Moeser, Oppenheim, and Siemerling). They occur more frequently in the basilar than in the vertebral artery. We may find a *true aneurism*, which may reach the size of a pigeon's egg or larger, or a simple aneurismal *dilatation;* the milder types are also not infrequently observed in the vertebral artery. Aneurisms are generally caused by specific or arteriosclerotic vascular disease; an embolus may also be the exciting agent. Traumata are not a great factor. The dilated vessel is generally also markedly tortuous, and various places on the pons and medulla oblongata are subjected to pressure. Evidence of this is generally found; the basal surface of the pons is notched; the olives, pyramids, etc., may also be atrophied or softened as a result of the compression, as in a case examined by myself, in which the basal area of the oblongata was completely softened. (Fig. 257.) Aneurisms may even press forward to the fourth ventricle. Slight atrophy of the olive was observed by me in several cases of slight arteriosclerosis of the vertebral.

Aneurisms of the vertebral and basilar arteries cause prodromes similar to those of cerebral arteriosclerosis, only that the occipital pain and impairment of the movements of the head are particularly pronounced. The bulbar symptom-complex manifests itself suddenly, or the disease develops *gradatim*. *Seizures* come on repeatedly during the malady, characterized by *anarthria, deglutitory paralysis, dyspnea, acceleration of the pulse*, and cardiac *arrhythmia*, and occasionally by a considerable *rise in temperature*. The bulbar symptoms gradually disappear, to come on again with a new attack. In the intervals also, phenomena remain which are due to an irritative or paralytic condition of one or several bulbar nerves, or to softening of the pons or of the oblongata or of the cerebellar peduncle (aneurisms of the basilar may also involve the cerebral peduncle). Rhythmical twitchings of the facial muscles and of the velum have been observed, more frequently paralysis of the facial, trigeminal, vago-accessorius, deafness, etc. The latter is present very frequently (Killian). The *alternating* character of these paralyses observed in some carefully studied cases is very characteristic. For instance, in one of my cases the vago-accessorius was paralyzed on one side, the hypoglossus on the other; in another, the velum was affected on one side, the facial upon the other side. The paralysis may be a simple or an atrophic one. The variable nature of the paralysis is probably due to the tortuous course of the blood-vessels. A paralysis of the extremities in the form of a general paraplegia, hemiplegia (hemiplegia alternans), or a paresis of the legs, is almost always present. Bladder and rectal disturbances are occasionally present.

FIG. 257.



Pressure-softening of the medulla oblongata from aneurismatic dilatation of the vertebral artery. (Drawn from a carmine specimen.)

A valuable sign of aneurism, to which Gerhardt particularly has called attention, is a vascular murmur on the back of the head. It has only been noticed in a few cases. A noteworthy symptom has been described by Hallopeau and Giraudau. In a patient who had a respiratory disturbance due to aneurism of the basilar artery, severe respiratory symptoms came on as soon as his head, which he held thrown back, was inclined forward; breathing stopped in expiration, to come on again only when the head was thrown back. In a case of Killian's, of aneurism of the posterior communicating artery, the head was also held backward.

The prognosis of these aneurisms is very unfavorable. The disease may last for months and even for years, but almost always ends in death either from softening of the cord or from rupture of the aneurism. The signs of rupture have been described above. The possibility of recovery is, however, not excluded, particularly when a specific basis is present. As lues is a very frequent cause, an antisyphilitic treatment should be instituted in every case. The treatment is in general similar to that for arteriosclerosis, for acute bulbar paralysis, and for cerebral aneurisms in general.

PSEUDOBULBAR PARALYSIS AND CEREBROBULBAR GLOSSOPHARYNGOLABIAL PARALYSIS.

The symptom-complex of glossopharyngolabial paralysis may be caused by a *cerebral disease*. The cortical centres of the facial, hypoglossal, and motor trigeminal are, as has already been mentioned, connected by conducting tracts with the nuclei of the medulla oblongata and the pons. A unilateral lesion of these centres and of the corticobulbar conducting tracts is naturally (with rare exceptions) not able to evoke symptoms of bulbar paralysis; it may induce a unilateral facial and hypoglossal paresis, while the masticatory, deglutitory, and laryngeal muscles are probably only paralyzed in bilateral disturbances of the cortical centres or conducting tracts. At any rate, cases have been observed in which multiple lesions of both hemispheres have produced a glossopharyngolabial paralysis, while the pons and medulla oblongata, as also the nerves originating here, have been found intact (Lepine, Joffroy, Barlow). In one of these observations, a cerebral multiple sclerosis formed the basis of the trouble (Jolly); in most of them, however, *atheroma* was the cause,—an atheroma which caused numerous apoplectic foci (softening, hemorrhage, cysts) in the cerebral white matter, more rarely in the cortex, in the central ganglia, particularly in the lenticular nucleus, and also in the internal and external capsules.

It is apparent that the symptoms of bulbar paralysis almost never come on here suddenly, but gradually, *in conjunction with many seizures*. The first attack excites a simple hemiplegia, which disappears up to a certain degree; a second may produce a repetition of the hemiplegia or it affects the other side of the body, and in the latter case symptoms of speech and deglutitory paralysis occur also. It may happen that only the bulbar nerves are paralyzed in the first attacks, while the body paralysis is absent or is present on one side only.

Pseudobulbar paralysis is characterized then by a glossolabiopharyngeal paralysis coming on in different attacks, which are followed mostly by bilateral, occasionally by unilateral hemiplegia. The paralyzed muscles retain their *normal volume* and their *normal electric excitability*. The *reflex excitability* is generally intact in the region innervated by the bulbar nerves. Bulbar laughing and crying (excessive lability) are very pronounced symptoms.

Siemerling and I have described spasms of crying and laughter with severe impairment of the circulation and respiration. Bechterew and Brissaud have observed the same. We presumed that it was a lesion of the centres or a break in the tracts which inhibited the bulbar centres. Bechterew gives the thalamus as the seat of the centres for such emotions; the tracts which conduct inhibiting impulses to these centres may be interrupted, or foci of irritation may be the cause. Brissaud thinks that these emotions are transmitted through a special tract in the anterior limb of the internal capsule, so that a break in the volitional tract (posterior limb of the internal capsule) causes all impulses to reach the anterior limb and to evoke an excess of emotion.

During these expressions of emotion, muscles contract which are no longer controlled by the will, particularly the facial musculature, so that closure of the lids may take place from reflex action and in emotion, but not volitionally. It appears that a bilateral break in continuity of the tracts passing from the cortical centres to the nuclei of the ocular muscles can excite certain disorders in ocular movement (an exterior pseudo-opththalmoplegia, according to Wernicke), a condition in which the patient has difficulty in voluntarily moving his eyes, while they will follow some object, and can be drawn to the sides by noises (Senator, Knies, Oppenheim).

It is important to know that multiple lesions of the cerebrum lead also to an *impairment of the mental condition*, so that *dementia, apathy, confusion*, and *conditions of excitement* are almost regular symptoms of this disease. Hemianopsia, aphasia, etc., are occasional symptoms. If aphasia is present, dysarthria can naturally be diagnosed only when the aphasia is incomplete. Pronounced disturbances of respiration, phonation, etc., are, as a rule, lacking, though the occurrence of the latter would be theoretically comprehensible, and has been observed in some cases.

Unmixed pseudobulbar paralysis is a rare disease. The cases examined by myself showed that in most of the cases included here the microscope revealed multiple lesions in the *pons* and *medulla oblongata*, so that we considered them to be cases of *cerebrobulbar* glossopharyngolabial paralysis. Senator, Otto, Jacobsohn, and others have furnished similar observations.

It is, therefore, though not a sharply circumscribed, still a, to a certain degree, typical clinical picture which we find in this disease. Its pathologic basis is a pronounced arteriosclerosis of some cerebral artery with its resulting conditions. The following alterations are observed in varied intensity: atheroma of the basal cerebral arteries and their branches, multiple foci of softening, hemorrhagic foci and apoplectic cysts of various sizes, ependymitis of the floor of the ventricle, chronic hydrocephalus, more rarely softening from compression of the parts of the pons and oblongata lying adjacent to the basal arteries and also of the cranial nerves. Naturally in such a severe vascular disease those parts of the brain which do not show material alterations suffer also.

The clinical picture can be sketched in a few words: clinical symptoms of general arteriosclerosis, eventually cardiac hypertrophy, nephritis, etc. The *mind* is affected. In advanced cases the patient is so helpless that he cannot associate with the rest of the world; attempts to talk cause spasms of hiccough, which may produce respiratory and circulatory disturbances.

Periodic conditions of frenzy and of mental confusion may also occur. *Anarthria* and *dysphagia* are present. Paresis of both lower branches of the facial, of the palatine velum, of the tongue, and of the muscles of phonation are found. The paralyzed muscles retain their normal volume and electric excitability; exceptions are rare and refer only to a circumscribed muscular area. Weakness of the masticatory muscles is occasionally present; more rarely, a masticatory spasm. *Respiratory disorders* are frequent,—attacks of dyspnea, Cheyne-Stokes breathing,—perhaps with increased temperature. These attacks appear spontaneously or upon attempts at movement and after pronounced emotion, or the spasms of hiccough pass into such an attack. It may last one or more hours. Increased pulse-rate and arrhythmia are also observed. The *optic nerves* are often involved; a pronounced neuritis or atrophy may even occur. The genesis of this optic affection is not yet clear. I ascribe it to pressure atrophy of the optic from the rigid carotid. Otto, however, under such conditions has found only slight alterations in the optic nerves. A simple or bilateral hemiplegia is generally present. The legs are often affected more than the arms. It is a spastic paralysis. Only rarely was an atrophy of some of the muscles of the extremities, due to small arteriosclerotic foci in the spinal cord, observed. The bladder and rectal functions may be impaired or normal. Marked anesthesia is rare. The disease generally develops after an apoplectic stroke. Such seizures are, as a rule, frequently repeated before the clinical picture has been completed. More rare than this step-like course is one which advances progressively after an acute beginning; but in this type, also, marked remissions and exacerbations occur.

Differentiation from progressive bulbar paralysis is generally not difficult. The apoplectic onset, the cerebral disturbances, the absence or incompleteness and asymmetry of the atrophy, the remittent course, etc., are sufficient to make a differential diagnosis. In one case of this kind, however, in which there was no history, I made a diagnosis of amyotrophic lateral sclerosis at first, until the dementia became distinctly noticeable. Involvement of the *optic*, *acoustic*, and of the sensory trigeminal always speaks against Duchenne's form. It differs from acute bulbar paralysis only in the mental disorder and the step-like development of the bulbar symptoms. It may be possible to distinguish nuclear from supranuclear forms by the absence of atrophy and by the intact reflex excitability in the latter. Trismus and mimic disturbances should occur, in general, only with the supranuclear forms. These movements cannot be valued with absolute confidence, however; for example, the condition of the reflexes is very inconstant.

In the last few years numerous cases of true pseudobulbar paralysis have been described (Lépine, Becker, Galavielle, Lereche, Brissaud, Münzer, Dejerine, Halipré), and it is positive that a disease-process confined to the cerebrum can evoke the symptom-complex of glossopharyngolabial paralysis. It is generally a bilateral affection of the motor zone, of the subcortical medullary layer, or of the deeper conducting tracts. Foci in the pyramidal tracts of the internal capsule on both sides have been noticed several times (Eisenlohr, Colman). Disturbances of phonation have also been observed.

French authors (Halipré, Brissaud) laid stress upon the fact that a bilateral disease of the lenticular nucleus (putamen) may cause the symptom-picture of glossopharyngolabial paralysis without a paralysis of the extremities; that this part of the brain, in this disease, is most often affected. They assume that there are special centres for the ordinary movements of deglutition, mastication, etc., and that the ansa lenticularis is connected with them. A bilateral lesion of these parts may impair, therefore, the above functions without the corresponding muscles being entirely withdrawn from the influence of the will. This is, however, only an hypothesis.

In some cases (Magnus, Kirchhof, Bamberger, Wallenberg) bulbar symptoms have been observed with unilateral cerebral lesions, though the examinations were not entirely free from objections, and also this phenomenon is so uncommon that we are forced to regard it as an individual aberration from the normal relationships of innervation.

The prognosis is unfavorable, though many years may elapse before death ensues, and the severe paralytic symptoms may even undergo marked improvement for a certain time.

INFANTILE FORM OF PSEUDOBULBAR PARALYSIS.—A particular form of cerebral bulbar paralysis was described by me in January, 1895; soon afterward Bouchaud published a similar case. Clinical observations of a similar nature were then given by König, Brauer, Ganghofner, and others. This type of glossopharyngolabial paralysis developed in connection with a cerebral infantile paralysis,—that is, an *infantile form of pseudobulbar paralysis.* It belongs to the category of diplegia, and is characterized by a bilateral paralysis or paresis (sometimes combined also with spastic—athetoid phenomena) of the labial, glossal, palatal, pharyngeal, and laryngeal muscles, with dysarthria, dysphagia, etc. In two of my cases trismus was present; in another, absolute mutism. Degenerative atrophy, fibrillary tremor, etc., were, of course, also absent in the muscles of the head. I was able to refer the disease to a bilateral lesion or malformation (*microgyria, porencephaly*) of the lower part of the central convolutions. Bouchaud found a similar condition.

Since then I have observed a number of similar cases. I have already referred, on page 541, to some peculiarities of these disorders.

The attempt of Schlesinger to include among the pseudobulbar paralyses the form of glossopharyngolabial paralysis produced by peripheral neural paralysis does not seem to me to be justifiable.

ASTHENIC BULBAR PARALYSIS (MYASTHENIA GRAVIS PSEUDOPARALYTICA, ASTHENIC PARALYSIS, BULBAR PARALYSIS WITHOUT ANY DISCOVERABLE ANATOMIC LESION).[1]

There are still a number of different forms of disease which are characterized by the symptom-complex of glossopharyngolabial paralysis. They include a number of clinical observations whose classification is at present impossible. There are, however, a number of cases which form a disease *sui generis*, and which, on account of their importance, deserve special consideration.

It first occurred to Erb that there was a form of bulbar paralysis which could be differentiated from the common form, particularly from the progressive, by its tendency to improvement, perhaps to recovery. In the cases described by him *ptosis* and *weakness of the masticatory and nuchal muscles* were the most prominent symptoms. *Atrophy* and decreased electric excitability were present.

At that time these cases could not be classified; now we are inclined to include them here.

In 1887 I was able, from a case observed clinically for a long time and carefully examined anatomically, to call attention to a new symptom-picture, in which bulbar phenomena took a prominent place. The most remarkable fact was that the careful microscopic examination of the nervous system gave a *negative* result. I was justified, therefore, in speaking of a *neurosis*, of a chronic, progressive lethal neurosis, which was particularly characterized by symptoms of glossopharyngolabial paralysis *without atrophy*. A case of bulbar paralysis without any anatomic find had already been described by Wilks, but in such an inadequate manner that a decision was not possible. My communication was followed by analogous and valuable observations of Eisenlohr and others. It was then particularly my assistant, Dr. Hoppe, who gathered together the cases, and, with one observed by myself and diagnosed *intra vitam*, made it evident that we were dealing with an *independent symptom-complex*, with a disease *sui generis*. The important signs, which I was able to demonstrate in my case, were *dysarthria, dysphagia, and masticatory weakness*, with corresponding paresis of the *labial, glossal, palatal, and masticatory muscles.* The *upper facial* was also involved; the closure of the lids was particularly weak. The paresis extended to the *muscles of the trunk and extremities;* there was present *marked weakness* in the arms and legs, also *dyspnea.* I was especially astonished by the absence of *atrophy* and of any *signs of electric degeneration.* These were also lacking when death came on after the disease had been present two and a quarter years. The pronounced *exhaustion* of the affected muscles and the tendency of the disease to *remissions*, the absence of anesthesia, and the marked pains were also characteristic. Attacks of severe apnea, tachycardia, and increased temperature appeared during the course of the disease.

---

[1] None of these names is at the same time definitive and comprehensive. I prefer them, however, to those which call the disease after one or more authors.

In some respects later observations have tended to enlarge the symptomatology, and, above all, to give important information concerning the *course.* In this respect the experiences of Goldflam are noteworthy, which teach that the patient may recover, that the phenomena reach their full development within a few weeks to several months, and that relapses, and, after some months, long remissions, may appear. This author has also made it probable that the cases described by Erb in 1878 belong here. It has also been shown that the *ocular muscles* are often involved: ptosis was observed several times. *Ophthalmoplegia* may indeed be the first symptom and dominate the clinical picture in some cases (Karplus). Recent observations have brought out more distinctly the muscular exhaustion. I observed in 1890, at the Charité, a young man affected with this disease, who was unable to take nourishment because, though he could chew the first bite, he tired out so easily that closure of the jaws could not be executed any longer with any force. The same exhaustion was noticeable in the muscles of the extremities: he could walk a few steps, and then the gait became wabbling, sluggish, and, after a time, the patient collapsed. *Electric stimulation of the muscles* had the same influence; in one sitting, in which the thoracic muscles were stimulated, a threatening *condition of suffocation* came on. The patient died. The autopsy showed an intact nervous system and normal musculature. Jolly made the microscopic examination, with a negative result. This author also studied carefully the above-mentioned anomalies of electric excitability, and grouped them under the name of "myasthenic reaction," and styled the disease "myasthenia gravis pseudoparalytica." Murri found that the muscles when exhausted by repeated faradization still reacted normally to the will. Later this *exhaustion* was made the cardinal symptom of the disease by Goldflam, Jolly, and Strümpell. I do not regard this as justifiable.

Since the first edition of this book numerous cases have been described (Shaw, Strümpell, Dreschfeld, Pineles, Murri, Grocco, Maier, Silbermark, Kalischer, Charcot and Marinesco, Karplus, Fajersztayn, Marina, Brissaud, Eulenburg, and others). The cases of Senator, Kalischer, Schlesinger, and Kojewnikoff cannot be positively included here. Wernicke seems to have been one of the first who observed this disease.

Grocco claims to have observed an exhaustion of the sphincter iridis.

If we group everything, we find that the following moments are characteristic of the disease:

(1) In addition to symptoms of bulbar paralysis, we find weakness of the muscles of the trunk and extremities; often also of the external ocular muscles (particularly ptosis).

(2) The affected muscles retain their normal volume and normal electric excitability.

(3) The weakness is subject to noticeable variation and combined with abnormal exhaustion.

(4) Sensory disturbances are lacking, except minor pains; the bladder and rectal functions are also unimpaired.

(5) The sensorium remains free.

(6) The anatomic examination is negative. Maier found, by the use of Marchi's method, alterations in the anterior roots. I regard it as questionable whether this observation, and also one by Marinesco and Widal, which showed changes in the ganglion cells, are of any importance.

The onset is acute, or, more often, subacute to chronic. It may progress acutely or chronically or step-like. Some cases (Bernhardt, Pineles) showed an intermittent periodic course. The process may commence in the bulbar muscles, in the ocular muscles, or in the muscles of the extremities. Youthful individuals were affected, as a rule.

We know nothing concerning the nature of the process. In a case which I observed with Bruns, *chlorosis* and a pronounced hereditary taint were present. I observed the disease once in a woman whose sister died from the same disease. A toxic genesis has been thought of.

In some cases an acute infectious affection had preceded the disease; in one patient a postdiphtheritic paralysis had been present years before.

Differential Diagnosis.—On account of the pronounced variations and remissions, it may be confused with *hysteria*. I observed it in one case combined with this psychoneurosis. It is more difficult to differentiate it from *polio-encephalomyelitis*. The most important differential points are the following: (1) Atrophy is absent; the electric excitability is normal, or only slightly diminished, or impaired in the sense of the myasthenic reaction. (2) Marked remissions and signs of exhaustion do not occur in polio-encephalomyelitis. (3) Optic neuritis may be combined with polio-encephalomyelitis; this is always absent in asthenic bulbar paralysis.

In the case observed by Jolly and myself there was much similarity to *muscular dystrophy*, which was also diagnosed by others, but the dystrophy and the corresponding alterations of electric excitability were absent.

The disease has also a superficial similarity to Gerlier's disease, *vertige paralysant*. (See the chapter on vertigo.)

It should be remembered that there is a form of Landry's paralysis (page 332) which commences in the muscles innervated from the medulla oblongata and results in death before the extremities are attacked by paralysis.

The prognosis is very unfavorable. Life is always in danger, though improvement and even recovery is not excluded.

The treatment requires great caution. I desire to warn particularly against *stimulating electric treatment*,—against the ordinary faradic or galvanic stimulation of the muscles which we are accustomed to use in bulbar paralysis. I regard these measures as *dangerous to life*, for the reasons already given. Central galvanization is permissible and advisable.

The most important requisite is the *avoidance* of all muscular exertion. The patient should keep in bed, should refrain from all muscular movements, and should speak as little as possible. In an advanced case, in which I was consulted, adherence to these measures produced marked improvement, but I was in error in one respect. I desired the patient to refrain from swallowing, and recommended feeding through an esophageal sound, without taking into consideration that the deglutitory movements of the patient would by its introduction be much increased. During the artificial feeding the patient died in an attack of choking. From this it can be seen that the pharyngeal catheter should only be used when it slides down easily and does not excite any spasms.

Tonics should be used. A careful diaphoretic treatment may also do good.

OPHTHALMOPLEGIA.

Paralysis of the ocular muscles occurs in various conditions and as a symptom of many diseases.

There are, however, disease conditions in which bilateral paralysis of the ocular muscles is the important element in the symptom-complex. Although it is hardly possible to separate the cases in which opththalmoplegia is recognized as being an independent disease from those in which it is only a component of the clinical picture, we are nevertheless justified in acknowledging its nosologic independence.

Ophthalmoplegia may develop *acutely*, subacutely, or *chronically*.

ACUTE OPHTHALMOPLEGIA is an affection which is chiefly due to infection and to intoxication. We have not as yet an adequate knowledge of its anatomic basis. There is no doubt that in many cases a hemorrhagic encephalitis—the acute hemorrhagic polio-encephalitis superior of Wernicke—is the cause (see page 529).

There are, however, toxic forms of acute ophthalmoplegia without an anatomic basis, and in such cases it may be difficult to decide whether a simple toxic paralysis or an encephalitis is present. This doubt holds particularly for those cases in which the ophthalmoplegia is a result of meat, fish, bologna, carbon dioxide, or other poisoning.

This affection may be also evoked by a simple traumatic hemorrhage into the central ventricles. This may be particularly true of traumatic late apoplexy. I, for instance, observed an ophthalmoplegia and paralysis of the masticatory muscles come on in a boy soon after a fall on the back of the head—a symptom-complex which is most easily explained by a hemorrhage into the central ventricles in the sense of Bollinger (see pages 470 and 501). There was also a hereditary predisposition present, as the mother, grandmother, and three aunts of the boy were afflicted with a congenital ocular disease (myopia, strabismus, cataract, and paralysis of ocular muscles).

The most important facts concerning the symptomatology of so-called nuclear ophthalmoplegia are given in another place, so that they can be omitted here.

It should be remembered that acute ophthalmoplegia may also be a result of peripheral neuritis (Dammron, Meyer, Dejerine), and that there are no absolute criteria enabling us to distinguish the neuritic from the nuclear form. The chapter on asthenic bulbar paralysis should be consulted for a differential diagnosis between it and this disease. An ophthalmoplegia without any corresponding anatomic lesion has been observed in Graves's disease (Bristowe, Warner, Ballet).

That nuclear ophthalmoplegia is often combined with bulbar symptoms has been sufficiently discussed in the preceding chapters.

The prognosis of acute ophthalmoplegia is doubtful. The form due to polio-encephalitis of *alcoholic* etiology seems to have the most unfavorable prognosis, though recovery is possible in this form also. If due to influenza, or following upon other infections and intoxications, it seems that recovery occurs in most cases.

CHRONIC OPHTHALMOPLEGIA, which should in most cases be also styled *progressive*, is rarely observed uncomplicated. A. v. Graefe described it first. The disease develops somewhat after the following manner. The patient becomes ill with ptosis or diplopia, only exceptionally with an associated paralysis of the ocular muscles. Gradually, in the course of some months or years, the paralysis extends to the ocular muscles *of both sides*, often in such a manner that the sphincter iridis and the muscle of accommodation, and also the levator palpebræ superioris, remain intact. This form, bilateral ophthalmoplegia exterior, shows plainly its nuclear origin. It is difficult to deduce such a paralysis from a peripheral process, while the construction of the oculomotor nuclei from cell-groups which probably represent the centres for individual ocular muscles (page 413) makes the nuclear genesis of an exterior ophthalmoplegia plain. It is also not uncommon for the internal ocular muscles to be involved from the beginning, or later. Monakow calls attention to the fact that in those cases in which the nuclear disease was detected anatomically, the sphincter iridis and the muscle of accommodation were particularly often involved.

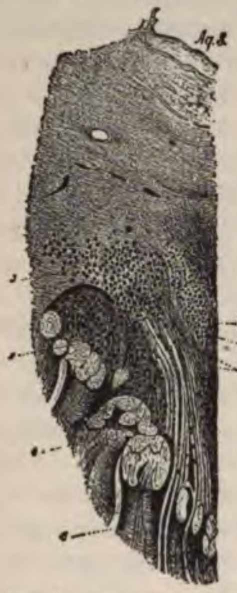
In advanced cases the eyeballs are immovable and the eyes look straight ahead or diverge slightly and do not follow any volitional impulses. Paralysis of the levator is in such cases also apt to be a partial one. In some cases the orbicularis palpebrarum was also involved. These phenomena may represent the whole disease, and after some months or years, when the total paralysis of the ocular muscles is complete, the process may come to a stand-still. Strümpell has described such a case in which the paralysis has been stationary for fifteen years.

The percentage of cases is larger, however, in which the chronic ophthalmoplegia is only the precursor or part of the symptom-complex of a complicated disease of the central nervous system: *tabes dorsalis, progressive paralysis, combined system disease of the spinal cord, multiple sclerosis, progressive bulbar paralysis*, and even *progressive muscular atrophy.*

Tabes dorsalis is particularly often combined with it, and five to seven years or longer may elapse before symptoms of the former manifest themselves. Its combination with progressive paralysis is also often observed; and it is noteworthy that the disease is particularly liable to combine with psychic disorders (Westphal, Siemerling). Those cases in which the disease descends from above downward are of particular interest,—*i.e.*, those in which the paralysis extends from the nuclei of the ocular nerves to those of the bulbar nerves. The facial on both sides is then affected first; next the glossal and palatal musculature, until finally marked signs of bulbar paralysis are present. The process may, however, commence at any place, and ascend or descend.

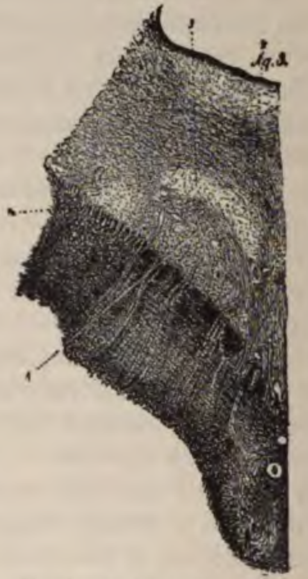
Finally, it should be remembered that ophthalmoplegia may be a congenital disease, or one acquired early upon the basis of an hereditary familial disease. Cases of this kind have been described by Gräfe, Mauthner, Hirschberg, Uhthoff, Moebius, Schapringer, Kunn, Heuck, and others. In such cases we find either a paralysis of some ocular muscles, particularly the levator palpebræ superioris, the abducens (bilateral congenital abducens paralysis may also combine itself with a corresponding facial paralysis), or a more or less complete ophthalmoplegia, which, however, spares the internal ocular muscles, particularly the sphincter iridis.

FIG. 258*a*.



Normal oculomotor nucleus. 1, ventral oculomotor nucleus; 2, median group of the third nerve; 3, lateral group of the third nerve; 4, posterior longitudinal fasciculus; 5 and 6, radicular fibres; 7, raphe.

FIG. 258*b*.



Atrophy of the oculomotor nucleus. 1, ventral oculomotor nucleus; 1*a*, dorsal oculomotor nucleus; 2 and 3, Westphal-Edinger's group.

(After Siemerling.)

We have but little definite knowledge concerning the anatomic basis of this congenital affection. Moebius assumes a defective development or an actual absence of the nuclear area (infantile nuclear atrophy). Heuck found the muscles absent or degenerated in one case with macroscopically intact nerves; he speaks of a primary muscle atrophy. Siemerling, on the contrary, detected a nuclear defect in a case of congenital ptosis. I have determined conclusively that a degeneration of the nuclei for the nerves to the ocular muscles may occur as a result of syphilis. The possibility of a hemorrhagic causation has also been spoken of.

The prognosis of chronic ophthalmoplegia is, on the whole, very unfavorable. The disease may disappear spontaneously or, if due to syphilis, from the treatment. It more often becomes stationary, but the signs of a cerebral or spinal disease generally follow, and the affected individual is threatened by this danger for years afterward.

Atrophy of the optic nerve is frequently combined with chronic ophthalmoplegia, be it that tabes dorsalis or progressive paralysis forms the original disease or that the optic atrophy is the complication.

Persistent headache and eye-pain were observed in some cases and supposed to be due to involvement of the cerebral trigeminal root (?).

The *anatomic nature* of *chronic* ophthalmoplegia consists always, or almost always (particularly from the examinations of Westphal and Siemerling[1]), in a *chronic inflammatory* or *degenerative process*, which is found on the floor of the third ventricle and the aqueduct of Sylvius, and which finally causes atrophy of the ganglion cells in the nerve nuclei (compare Fig. 258). It seems to be a primary disease of the ganglion cells, which then involves the entire nucleo-peripheral neuron. The roots, as well as the peripheral nerves and muscles, are degenerated in accordance with the involvement of their trophic centres.

With these alterations there are eventually combined those of tabes dorsalis, paralytic dementia, progressive bulbar paralysis, etc.; or these affections are only partly developed.

Treatment.—In acute cases, an antiphlogistic or diaphoretic treatment is in place,—ice-bag, bloodletting in the temporal region, purgatives, and sweat-baths. I have in some cases seen distinct improvement from the latter. If lues is the cause, an antisyphilitic treatment should be instituted. The same is indicated in chronic cases also, if syphilis had been present. In addition, the causative factors must be treated in all cases. Electric treatment has no noticeable effect, though galvanic treatment is recommended by some. In two of my cases, improvement occurred during a stay at Oeynhausen.

---

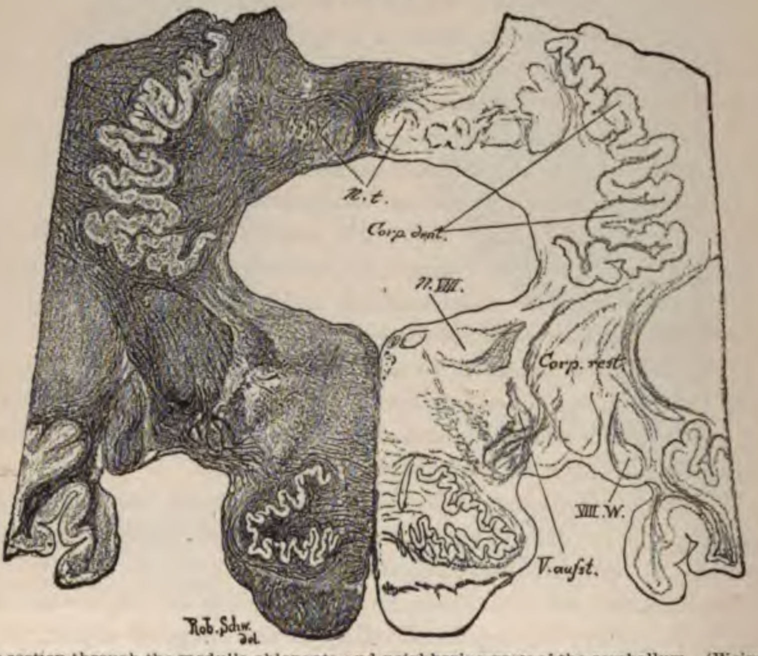
[1] Hutchinson, and Gowers before him, later, Boettiger, myself, Boedeker, Pacetti, Marina, Zeri, Cassirer and Schiff, and others, have made similar observations; and Siemerling and Boedeker have made new statistics lately from a large amount of material.

DISEASES OF THE CEREBELLUM.

The morphologic division of the cerebellum into a number of segments with special names has until now not been of much pathologic importance. Our knowledge of the functions of the whole organ is also still far from complete. Concerning the anatomic relations, page 407 and those following should be consulted.

In many respects, the results of experimental examinations and of clinical pathology agree. The latter are for us the most important.

FIG. 259.



Cross-section through the medulla oblongata and neighboring parts of the cerebellum. (Weigert's stain.)

The cerebellum has no connection with the special senses or with conscious sensation. It has a particular influence upon muscular movements. The chief disturbance noticed in cerebellar diseases is a motor incoördination. It is especially noticeable in standing and walking. Upon standing the patient sways; he stands with legs spread apart. The uncertainty is recognized from the balancing movements, which are due to contraction of the extensors of the foot and toes. The swaying, as a rule, is not increased upon closure of the eyes. The gait resembles that of a drunken person. The legs are more affected than the arms. Upon simple movements of the limbs,—*e.g.*, upon lifting the leg from a recumbent position,—ataxia is generally not present, though cerebellar diseases may also excite static ataxia. Nothnagel regards incoördination as arising from the middle lobe; he is, however, probably only so far right, in that many cerebellar tracts decussate here, so that lesions of this area influence the functions of both hemispheres.

Diseases which are confined to the cerebellar hemispheres may run a latent course. Irritating processes, which have their seat in the cerebellar hemispheres or in the middle cerebellar peduncle, may cause *involuntary movements and positions*. Of these the most characteristic is rotation around the axis of the body, with a tendency to fall to one side. Abnormal positions of the eyes are also found with these conditions.

Paralysis is often observed in cerebellar disease, either *unilateral or crossed hemiplegia*. It is probable that this is not a direct result of the cerebellar lesion, as it almost exclusively occurs in diseases causing compression. There is hardly any doubt that *compression of the pyramidal tract* in its course through the pons and oblongata is the cause of this paralysis, and that it depends upon the location of the compression,—viz., before or after the pyramidal decussation, whether the hemiplegia develops upon the same or upon the opposite side to that of the cerebellar disease. Luciani and some clinical observers ascribe to the cerebellum some influence upon the strength that can be exerted upon the homolateral side.

Motor symptoms of irritation (general, unilateral convulsions, tremor, clonic twitchings) are also probably not the result of the cerebellar disease but dependent upon irritation of the motor areas in the neighborhood.

This is probably also true for the *vomiting*, which can doubtless be traced to involvement of the medulla oblongata, as also for the symptoms of irritation and paralysis in the region of the motor cranial nerves. There is nothing definite to be said of the *nystagmus* which occurs in diseases of the cerebellum; this is probably also a pressure symptom. The same is doubtless true of the intentional tremor, which is repeatedly observed here.

Psychic disorders have been often observed in persistent diseases of the cerebellum, though complicating cerebral diseases, as hydrocephalus, etc., were probably present.

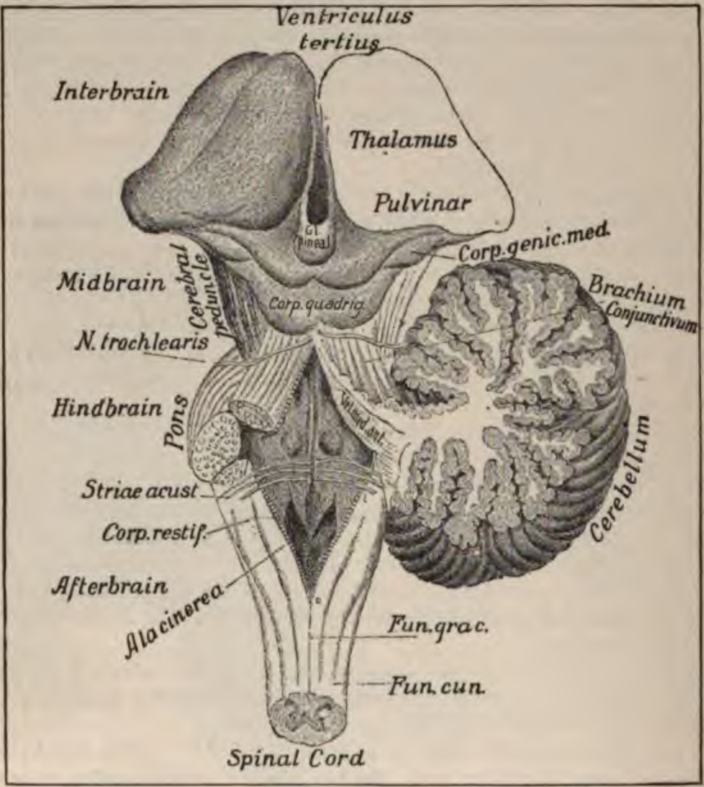
The *speech disturbances* which occur in cerebellar diseases, described as dysarthria or scanning speech, or as ataxic speech, might be due to action of the process upon the medulla oblongata, or to an independent disease of it. It is not improbable that the co-ordinating influence of the cerebellum also extends to the articulatory muscles, so that disease of this organ causes an incoördination of the movements of speech. We have then as direct cerebellar symptoms only the *incoördination*, the *vertigo*, and probably the *speech disorder*.

Schiff and others, and recently Jackson and Russel, have ascribed a special influence of the cerebellum upon the truncal muscles; this is correct in so far that in the maintenance of co-ordination it takes a prominent part.

How does the cerebellum fulfil its co-ordinating duties? With Bechterew, Bruns attempted to make this action clear. The cerebellum receives impulses from the skin, muscles, joints, semicircular canals, and other peripheral parts, which regulate its actions. The centripetal paths which are in question are the direct cerebellar tract, Gowers's tract, the posterior-column-cerebellar tracts, the vestibular nerve, and perhaps also the central tegmentary and the cerebellar olivary tracts (compare page 405 and following), also the fibres traversing the restiform body. The cerebellum must also act upon the musculature of the trunk and of the extremities, either directly or through the motor centres, or in both ways. A (hypothetical) tract passing through the middle cerebellar peduncle to the pons and from there to the fundamental fasciculus of the anterolateral tracts, has been given as a direct motor tract; there are also tracts in the upper cerebellar peduncle which connect the cerebellum, through the optic thalamus, etc., with the cerebrum. Even though these are largely centripetal paths, it is not excluded that stimulations of the motor centres may in this way reach the cerebrum. Finally, we can imagine that the cerebrum may affect the mechanism of the cerebellum. The cortico-cerebro-pontine tract, which connects the frontal lobes with the contralateral cerebellar hemisphere, probably serves to create such relations. We find motor centres for the truncal muscles in the frontal lobes. While these centres, as Bruns showed, govern *voluntary* movements for the maintenance of co-ordination, the cerebellum exercises its influence reflexly and automatically upon co-ordination; but these two central apparatuses may act upon each other.

We can then understand how disturbances of co-ordination—*cerebellar ataxia*—are primarily caused by cerebellar diseases, but also by affections of the frontal brain, the cerebellar peduncle, and of all the tracts coming towards and leading to the cerebellum. The diagnosis can only be made from the accompanying phenomena, as well as from the intensity and timely appearance of the cerebellar ataxia.

FIG. 260.



Hindbrain and afterbrain, opened by removal of the roof. Cerebellar peduncle visible. (After Edinger.)

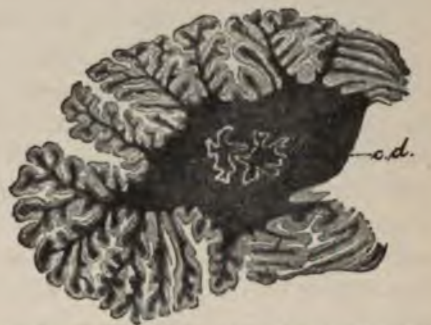
This conception also enables us to understand how disease of the cerebellum, when it develops gradually or affects only a part, can, to a certain extent, be hidden by the cerebral symptoms. In all those parts of the brain, through lesion of which an ataxia similar to cerebellar ataxia may be evoked, there are tracts which have a direct anatomic connection with those of the cerebellum.

Diseases of the cerebellum are of various types, and for the most part have been described in other parts of this work. We can have hemorrhage, softening, inflammation, and, more often, abscess and tumors of the cerebellum. *Atrophy* and sclerosis of the cerebellum have not yet been discussed.

The older observations of this kind were described by Nothnagel and Hitzig; we have numerous recent observations, the most important being those of Claus, Fraser, Sepilli, Hammarberg, Moeli, Nonne, Menzel, Schultze, Arndt, Cramer, and Spiller.

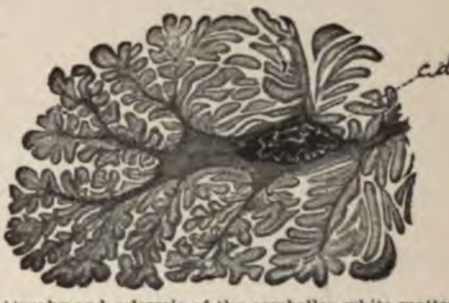
We find either a *congenital* smallness or a congenital dysmorphosis, in which some lobules, or a whole hemisphere, etc., may be missing, or are only rudimentary, or an *acquired* condition which results in shrivelling, induration, and atrophy of the cerebellum or of some of its parts. They are then either developmental disturbances or focal diseases occurring in fetal or extra-uterine life. Some are of vascular origin (inflammations, softening, perhaps also hemorrhages) and some of meningeal origin.

FIG. 261*a*.



Normal cerebellar hemisphere upon cross-section. *c.d.*, corpus dentatum.

FIG. 261*b*.



Atrophy and sclerosis of the cerebellar white matter. (Compare with 261*a*.) (Pal's stain.)

There is no doubt, as I can see from my own observations and from the literature, that there is a *cerebellar form of infantile palsy*, in which the affections described (on page 533 and following) as causing cerebral infantile palsy can also localize themselves in the cerebellum. In some of the cases the disease comes on acutely under the picture of a severe cerebral disease to retrogress later to a condition of stationary paralysis. It has often been noticed that children in the first few years after the onset of the disease are not able to walk, or can only crawl on all fours.

In the case observed by myself and Arndt the disease was acquired in later life, and was apparently due to atheroma. The process caused here a sclerosis and atrophy of the corpus dentatum (compare Fig. 261*b*).

The *phenomena* which have been observed in cases of cerebellar atrophy were not the same in different cases, but generally consisted of the following symptoms: *defective mental development, reeling gait* (*cerebellar ataxia*), *vertigo, speech defects,* which have been described as dysarthria and scanning speech, or as an intentional tremor, or an ataxia of the muscles of speech; increased knee-reflexes were mentioned several times; and *epileptic attacks, paralysis of the ocular muscles,* or an abnormal position of the eyes, several times also a *tremor,* which was not described very clearly, have been noticed. According to the views expressed before, only the cerebellar ataxia and the vertigo, perhaps also the speech disturbances,(?) are due to the cerebellar process. The hydrocephalus, which has several times been found (Kirchhoff, Sommer), is probably responsible for the psychic disturbance.

This atrophy has also been detected in several cases which were very similar to Friedreich's disease. Nonne, for instance, described a familial disease, due, as he was able to determine, to an abnormal smallness of the whole central nervous system. It developed in three brothers at the time of puberty, or later in life, and ran a chronic course. The symptoms were a loud and explosive speech, nystagmus, imbecility, paralysis of ocular muscles, and simple optic atrophy, disturbances of co-ordination, and increased knee-reflexes. All of these symptoms, it is needless to say, cannot be referred to an abnormal smallness of the cerebellum. Marie has described a disease, which he calls cerebellar hereditary ataxia (hérédoataxie cérébelleuse), which he regards as being due to hereditary atrophy of the cerebellum.

SECTION IV.

THE NEUROSES.

HYSTERIA.

UNTIL recently the knowledge of this disease, notwithstanding its frequency, has been shrouded in darkness, errors having been made in the significance of individual phenomena, as well as in its general conception. This is due to the fact that it is a *psychosis*, which does not express itself by disorders of the intellect, but in defects of character and in emotional disturbances, whose real nature is hidden under an almost unlimited and varied number of *physical* symptoms of disease.

Women are more often attacked than men. The figures of Briquet, that fifty men become hysteric to one thousand women, must, in the light of later experience, be modified to one to ten or one to six; according to Gilles de la Tourette and Pitres, one to two. We do not, however, agree with the latter.

The disease generally begins at or about the age of puberty and afterward up to the thirtieth year. Early childhood is, however, not exempt. I have seen it often in children of from four to eight years of age, as often in boys as in girls. I have also repeatedly observed hysteric symptoms in children of from two to three years of age. (See chapter on infantile eclampsia.) The disease is very rarely observed in those who have reached the climacteric. Some speak, however, of a senile hysteria.

Although no race and no nation are spared, there is some variability as to its occurrence among them. It is particularly common in France; the Anglo-Saxon is much less easily influenced. The Jews are especially liable to hysteria.

Causes.—There is often a *congenital* or *hereditary* predisposition. Direct heredity is very common. The children of an hysteric mother are either originally hysteric or are predisposed to it, so that every shock to the nervous system may be an exciting cause. It may also be inherited from ascendants who had some other nervous disorder. It is more rare for the father to transmit it. I treated four children of an hysteric father, all of whom were attacked by hysteria when six to seven years old, each with a different clinical picture. Bernutz reports a similar experience. The hereditary influences are increased when both parents are neurotic. Consanguinity and twin-births are also predisposing factors. *Alcoholism* in the parents may predispose to it. This form shows itself at an early age in an *irritable nervous temperament.* The more excitable an individual is, the more liable he is to become hysteric.

Charcot and his school considered hysteria to be always inherited, all other factors being merely occasional or exciting causes, which arouse the latent disease, but which are unable to produce it. This is, however, too general a statement. It is certain, however, that all other movements are more effective and operative provided a predisposition is present. Among the other causes, the *emotions* are particularly provocative. A severe emotional shock (terror, anxiety) can, without doubt, produce hysteria: it is generally long-continued, repeated painful agitation and more often the pain which man gives to man than those sufferings which are the result of accident.

The nerve-shattering influence of psychic traumata explains to some extent the reason that the neuroses and psychoses, particularly hysteria, are so frequent among the Jewish people. Other factors, however, as the frequency of intermarriage, their defective physical development, etc., are also in play.

The paralyses resulting after *lightning-strokes* and *earthquakes* are partly a result of *psychic excitation,* and may be hysteric.[1] *Mental* exertion cannot of itself produce hysteria, but may act as an exciting agent. Excessive mental work, combined with continued excitement, may indeed be a cause of this disease; but here, also, the mental emotion is probably the active agent.

Irritative impulses may initiate in predisposed individuals hysteric symptoms, especially symptoms of irritability. This occurs especially in cases of hysteria in childhood; it also explains the epidemics of hysteria, which have become more rare in our time, but which in former centuries were so common.

All diseases which cause loss of strength and increased secretion may evoke hysteria. The relations of the uterus to this disease have been greatly overvalued. Diseases of the sexual organs, it is true, influence detrimentally the psychic life: women afflicted with uterine trouble are often unfruitful, their sexual life is more or less injured, their marriages unhappy, and these factors are the source from which hysteria may originate. *Masturbation* is an important etiologic factor. Freud, however, goes too far in considering "sexual traumata" of early childhood, arising in genital irritation, to be the specific cause of hysteria.

I have often observed hysteria resulting from *castration,* as have others; a predisposition, however, generally existed. The troubles of the *climacteric* are partly due to hysteria and neurasthenia; disorders of the vasomotor nervous apparatus are especially at fault here.

---

[1] In an earthquake which I went through, I observed in many of my companions, as an effect of the psychic excitement, localized muscular twitchings, especially facial spasm.

Symptoms very similar to those of hysteria, and, according to Charcot, identical with them, may arise from chronic *alcoholism* and the chronic intoxications of *lead, carbon dioxide,* and *mercury.* Syphilis and the acute infectious diseases may also be provocative agents.

Every *trauma,* when combined with psychic excitement, may produce hysteria. On the other hand, as a result of cranial injuries or general or local concussions, we often observe symptom-complexes develop in which hysteric symptoms take a prominent part. (See chapter on traumatic neuroses.)

Nature of the Disease.—The basis of hysteria is the abnormal irritability and the *sudden variations in disposition.* The emotions are excited by slight agents, are very pronounced, vary easily in form, and have an abnormal action upon the motor, sensory, vasomotor, and secretory functions,—*e.g.,* the abnormal emotional life does not alone show itself in the *lack of desire* which accompanies psychic actions, but also in *physical phenomena,* in *pathologic increase of normal actions;* laughing and crying become spasmodic; the transient inhibition of function, which in normal persons is produced by fright, becomes paralysis; the blushing, which contempt, anger, shame, etc., produce in normal individuals, may here occur at every slight emotion, and in increased intensity, etc.

Another important factor is the *increased power of the imagination* (suggestibility). Healthy minds have also the power, without adequate stimulation, of producing mental conceptions and sensations. But in the hysteric person this power is so much enhanced that hallucinations occur, and previous happenings in life are not only remembered but relived. The imagination may in the mentally sound also excite sensations and phenomena which normally are caused by sense-stimulation (salivary flow in thinking of a savory dish, vomiting and distress from the idea that something disagreeable had been ingested, etc.). This *influence of the imagination* is increased in hysteria and altered, so that the conception of a paralysis may produce a paralysis, etc. All hysteric phenomena are *changeable;* they may come on suddenly, and as quickly disappear; and these changes may almost always be referred to psychic influences. The results of treatment obtained by a physician are also, to a great extent, the product of psychic influences.

Breuer and Freud call attention to the fact that psychic traumata (anger, vexation, fright, etc.) which do not produce a psychic disorder exert to a certain extent a latent influence.

Symptomatology.—*Psychic Condition.*—An hysteric individual is very irritable. The slightest circumstances produce marked excitation which, according to the cause, shows itself in depression, anger, vexation, terror, anxiety, etc. The patient's frame of mind may undergo rapid variations without there being any known reasons for them. The remembrances of previous events are very vivid, evoke feelings of dis taste, which may exacerbate from time to time to outbursts of pain or of anger, and even produce conditions of dream-like confusion, in which, mistaking his surroundings or situation, and without realizing that they are not actual occurrences of the present, the patient re-lives either past events or dreams.

The ability for inhibition and for repression of the emotions is lost. Notwithstanding the fact that the *will-power* for certain (physical) actions is injured, we cannot speak of a general weakness of the will. Hysteric patients more often develop an astonishing amount of energy in order to accomplish a definite purpose.

The *mental power* is not weakened. Hysteria is more often found in intelligent than in ignorant persons; it may, however, attack weak-minded and degenerated individuals or be combined with conditions which lead to feeble-mindedness; this is, in such cases, however, always a combination of independent diseases. Though the acts of an hysteric individual often appear unreasonable and motiveless, it is due to the fact that the feelings of the individual, abnormally increased in intensity, force him into committing sudden (explosive) actions. This explains the incongruities in the character of an hysteric, in whom we often find peculiarities which stand in marked contrast to these pathologic actions and expressions. The rapid variability in symptoms does not permit any stability of character—the patient's mental nature is not a unit, but consists to a certain extent of two or more. Particularly, however, the false opinion which the outside world and, unfortunately, also often the physician hold in regard to this disease, produces a bitterness of spirit and often an effort to show the well-known symptoms of hysteria more distinctly and vividly than is natural.

The few cases in which hysteric patients injured themselves and ascribed their injuries to results of the disease do not alone denote an inborn desire for deceit and simulation, as was formerly wrongly thought, but indicate the severe psychic disorder which occasionally occurs in the course of the disease. Hysteric prevarication is almost always pathologic, and may often be referred to transformation of the memory.

An hysteric (or, better, a psychopathologic) predisposition may be betrayed early in life through *abnormal avarice.* The *memory* is generally intact, though now and then the attention is so concentrated upon the inner self that many impressions are not received or fixed in the mind. Janet speaks of a "contraction of the conscious field" which makes it impossible for the hysteric to receive in his consciousness a series of impressions and associate them with his "ego." The attacks may also leave gaps in his memory, so that the remembrance of a certain time is completely or partially blotted out (retrograde amnesia). The ability to retain fresh impressions in the memory is less often lost.

Examining more closely the psychic disturbances which are exhibited in *paroxysms*, we find:

1. *Conditions of Fear.*—Almost all hysteric patients complain of a sudden, severe sensation of fear combined with a feeling of pressure in the precordial region, or with a palpitation of the heart, and these phenomena may be the chief subjective symptoms. This fear is generally unaccompanied by definite conceptions and phobias.

2. *Hallucinatory Delirium.*—This is a condition of dream-like confusion of sudden onset in which the patient is under the influence of hallucinations, especially frightful visions, with corresponding movements of the body. The face expresses fear, amazement, and anger; the patients strike out wildly around them; run as if from an enemy, secrete themselves in a corner, etc., and appear to be entirely removed from their surroundings. Often, however, they may be slightly influenced, shrink away, or strike at you when they are approached, or may be quieted for a few moments. Occasionally, by acting energetically in opposition, or by strong stimulation of the skin (sprinkling with cold water), they may be made conscious of their surroundings.

These attacks, which last some hours, rarely days, I have seen also in hysteric children, especially in boys, and once in a two-year old child of marked neuropathic taint. This delirium occurs isolated or as a concomitant phenomenon of an attack of spasm.

3. *Somnambulistic and Hypnoid Conditions.*—On account of their close relationship with the spasms of hysteria, these conditions will be discussed in connection with them.

A pronounced *mental disorder* of the character of melancholia, mania, paranoia, or folie raisonnante rarely develops during the course of a hysteric attack. This, if it does occur, does not indicate that it is a case of pure hysteria, but rather hysteria in combination with another psychosis, even though it has hysteric earmarks, and gives it a peculiar nature. The compulsive ideas do not belong to the symptom-complex of hysteria any more than do dipsomania, agoraphobia, and allied disturbances. Their occurrence only shows that the cause of the hysteria may produce other diseased mental conditions.

Disorders of Sensation.—*Pain* is always present. It may be located in any part of the body, and may have the character of neuralgia, migraine, angina pectoris, rheumatism, or any other form of pain. *Headache* is very common. It does not always have peculiar characteristics which enable us to recognize its hysteric nature. There are, however, certain forms and accessory symptoms which are of value in the making of a diagnosis. It is very often a *hyperesthesia of the skin of the head.* The patient complains that he can hardly comb his hair on account of the distress it causes, and that the slightest touch to his head is painful.

Clavus is a well-known type of hysteric headache. It is a pain felt upon a circumscribed area of the side or the vertex of the head, which persists for hours or days, and may be accompanied by vertigo and nausea and vomiting.

Ordinary *hemicrania* is also very common in hysteria, and is the form which has a tendency to last for weeks or months. (See Hemicrania.)

Occipital pains are often present. It is either a pain in the nuchal region or in the back, passing up to the occiput, and extending from here to the eyes. All the types of headache, as are all the symptoms, are under the mental influence of the patient, and, therefore, the subjective element is prominent.

The pains may be in the trigeminal region and be similar to those of trigeminal neuralgia. The differential disgnosis between true and hysteric neuralgia has been given under neuralgia. The pains in the back are almost as frequent as headache, and are found along the entire back or only in a circumscribed area. They are often characterized as being of a burning character.

Pains in the intercostal region, particularly the left, corresponding to the type of an intercostal neuralgia, severe pains in the coccygeal region, combined with pronounced sensitiveness to touch and with contraction of the muscles of this region (coccygodynia or sacrodynia), and pains which appear to be located in the muscles, fasciæ, or periosteum, are also frequent. There is also a hysteric form of breast-pain, its seat being in the mammæ (*mastodynia*). It may be very stubborn, and be combined with hyperesthesia of the skin, reddening, edema, general and circumscribed swelling of the mammary glands, and even with ulceration of the skin. This disease, described by Charcot and Gilles de la Tourette as "sein hystérique," has, as has also simple mastodynia, given cause for amputation of the mammary gland.

These pains are not of local but of central origin, and may be classed as hallucinations of pain,—*i.e.*, as direct excitations of the pain-perceptive centres, though they are often due to slight peripheral stimulation.

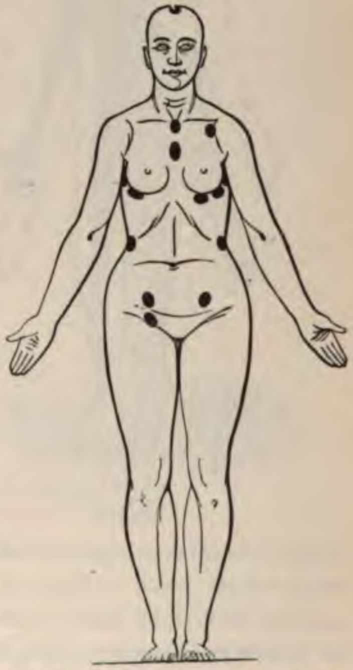
Paresthesias are also common. Formication and numbness are especially frequent. *Hyperesthesia* is rarely complete; the sensitiveness being mostly limited to *circumscribed* areas, small patches of the skin or corresponding parts of the deeper structures (muscles, fascia, abdominal organs), or to an extremity or section of a limb. For instance, the skin of a joint which is contracted will be found hyperesthetic. A *hyperesthesia plantaris* is also a symptom of hysteria. These hyperesthesias are often combined with the pains and are difficult to separate from them.

The hyperesthetic areas are at times identical with the *hysterogenic zones*. These are regions which are at times the seat of spontaneous pains, at other times react to pressure, so that an attack (cramps, paroxysms of pain, etc.) is produced, preceded by some *aura* commencing in this region. At times, also, pressure upon these or other points (hysterofrenatic zones) may abort an attack. These hysterogenic or spasmogenetic zones are found all over the body (Fig. 262), but deserve special attention in the *ovarian* region, because they present here most prominently their typical characteristics. Whether the ovary is the starting-point of the trouble, and whether the hand pressed into the lower abdomen must reach the ovary to produce or stop an attack, is doubtful. The zone generally lies higher than the ovary; pressure on this place in hysteric men is also successful in evoking or aborting attacks. The region of the apex-beat is a frequent location for this hyperesthesia, also the stomach.

Hysteric backache is generally combined with hyperesthesia of the skin over the spinous processes, which is more severe than that occurring in spinal diseases. The psychic nature of this hyperesthesia is easily recognized by the fact that when the attention is distracted it does not appear.

The special senses are also hyperesthetic. An excessive sensitiveness to light, a noticeable increase in the ability to recognize faces, and phenomena in the other special organs, combined with idiosyncrasies (abnormal distaste for certain taste- or smell-impressions, excessive desire for others) and paresthesias—flicker-

FIG. 262.



Hysterogenic zones on the anterior surface of the body. (After Bourneville and Regnard.)

ing before the eyes, buzzing in the ears, etc.,—belong to the common phenomena of hysteria. Visual hyperesthesia may be combined with an increased visual field (Freund), but this occurs very rarely indeed.

The *anesthesia* of hysteria is an important diagnostic point, as an objective decrease of sensation is found in most cases. Pitres missed it in only two of forty cases. Hysteric anesthesia is characteristic (1) in the manner of its extension, (2) in its close connection with disturbances of the organs of sense, (3) in its psychic nature and its reaction to external influences.

It never confines itself to the area innervated by a particular nerve or nerve-plexus. It is often limited to one-half of the body. This *hemianesthesia* is sharply outlined by the median line (Fig. 263); it seldom extends over it or falls short of it. It extends still more rarely over the whole body-surface. In both cases we find, as a rule, circumscribed areas with normal or increased sensation. The deeper structures (periosteum, nerves, joints) also take part in the anesthesia.

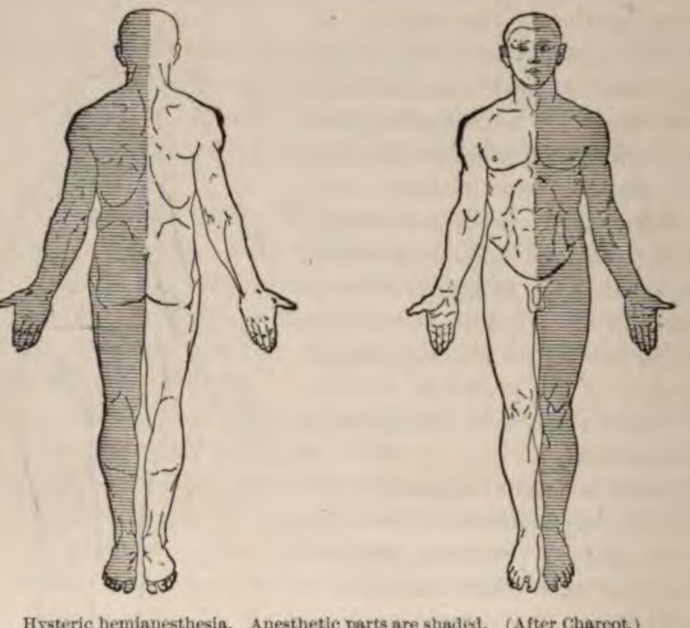


FIG. 263*a*. FIG. 263*b*.

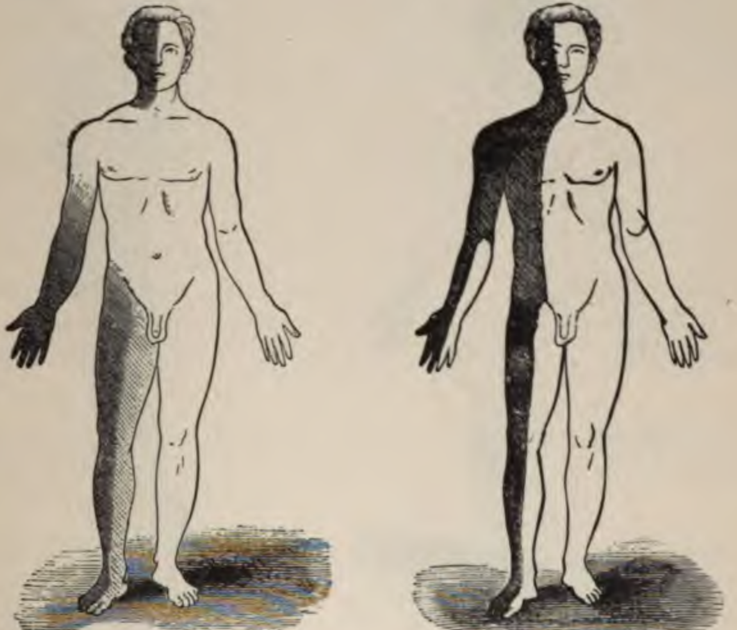
Hysteric hemianesthesia. Anesthetic parts are shaded. (After Charcot.)

The loss of sensation is often limited to definite parts and sections of the body, and presents then a peculiar extension of the anesthesia, the areas involved being sharply demarcated, and the anesthetic region being of a stocking, glove, or drawer's-leg shape (Figs. 264 to 267).

Only in syringomyelia do we find a similar mode of extension, though differences between these two may also be noticed (see page 229).

The anesthesia may also limit itself to the skin of the head and frontal region (Fig. 266), and divide it from the rest of the head. Or it affects the arm to the shoulder-joint; it embraces the upper arm and neighboring part of the trunk in a half-vest-like form or the hand and forearm like a glove, etc. As a rule, it is demarcated by lines which are vertical to the longitudinal axis of the extremity (amputation lines). Parts of the body are involved that are thought by the laity to represent a unit, as the arm, leg, hand, etc. This form of geometrical or segmentary extension is especially the case in paralyzed limbs with contracture. An anesthesia occurring in the form of islets is more rare.

FIG. 264. FIG. 265.



Hysteric anesthesia. (After Thomsen.)

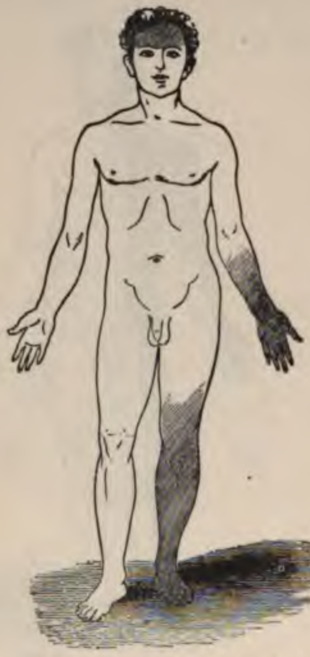
Disorders of the special senses are generally combined with the sensory disorders. This is most marked in hysteric (sensory or mixed) hemianesthesia. It is generally accompanied by decrease or loss of the senses of smell, hearing, taste, and sight, on the same side.

The ocular disturbance which, of these phenomena, is the most important, is rarely a decrease of central acuity of vision; it is often a *concentric contraction of the visual field*, which must be tested with the perimeter. The concentration for blue is often more marked than for red, while it is present in a typical order for white and for other colors.

Achromatopsia and *dyschromatopsia* are less frequent,—*i.e.*, the colors are not recognized at all, or are confused with each other (especially red and green).

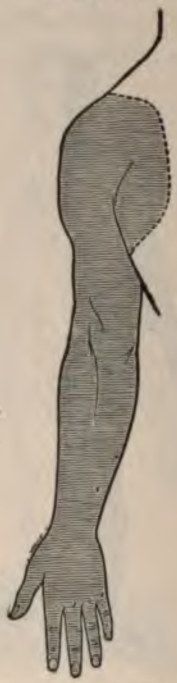
The concentrically contracted visual field is usually found on both sides, but is more pronounced on the anesthetic side. A complete amaurosis is not common, and cases of *total bilateral hysteric blindness* are observed rarely. Decrease in taste- and smell-perceptions need not be for all qualities of these sensations; it also happens that only a certain part of the mucous membrane for taste is affected.

FIG. 266.



Hysteric anesthesia. The shaded parts are the anesthetic regions. (After Thomsen.)

FIG. 267.



Hysteric anesthesia. (After Charcot.)

The decreased hearing shows its nervous origin by the decrease or absence of osseous conduction, which is at times more marked for high tones than for lower ones. The involvement of the senses may not be complete or may be absent, and often one or more sense-organs are spared.

In unilateral cutaneous anesthesia the special senses may be impaired on both sides (this is the rule for vision). It is, however, rare for a kind of decussation in this sense to be present. Sensory disturbances are also not always accompanied by anomalies of cutaneous sensation. The anesthesia may extend to the bladder and rectal mucous membranes (always on both sides), and produce secondary disorders of these organs. A hemianesthesia may also be found in the vagina (Briquet).

Character of the Anesthesia.—All sensory qualities may be affected. *Analgesia* with intact tactile sensation, or *analgesia with thermanesthesia*, may exist alone; an isolated thermanesthesia is rare. Further, we frequently find, perhaps in most cases, not a complete, but a partial loss of sensation, a hypesthesia not an anesthesia, which may be so slight that it can only be detected by tests on symmetrical parts of the two sides of the body with stimulation of equal degree. It should be remembered that analgesia to prickings of a pin does not always indicate complete loss of pain in the use of a faradic brush.

In complete anesthesia a fold of the skin may be pierced, or the needle may penetrate to the periosteum without being felt. Pinching of the muscles and pressure upon the nerve-trunks are likewise not painful. The patient cannot locate the position of his limbs, and he may, on account of loss of sensation, be unable to execute any movements with closed eyes. Closure of the eyes may at times in such cases produce a condition similar to sleep (Strümpell) which is probably of an hypnotic nature.

We denote by *haphalgesia* the phenomenon in which ordinary touching of the skin with objects, which at other times produce only tactile or non-painful sensations, occasions severe pains.

The *reflexes* do not present a constant relationship with the hysteric sensory disturbances. The peripheral reflexes corresponding to the hypesthesia or anesthesia are decreased or lost, though complete absence of the corneal reflex is infrequent. The pupillary reflex to light, the cremasteric, and the inconstant abdominal reflex are uninfluenced. Where only analgesia exists the reflexes may react normally to tactile stimulation; where only hypalgesia is present, severe pain may still excite reflex action.

The skin reflexes may be unobtainable upon an examination for which the patient is prepared, while they may be elicited through sudden and unexpected stimulations. Fright may be a factor in such cases.

It is true, however, that hysteric individuals often feel and experience sensations, though they are not aware of it. That an hysteric person with a unilateral amaurosis may frequently still see with the blind eye may be proven by the use of a prism or stereoscope. The ability for orientation, despite a marked contraction of the visual field, is generally only slightly impaired. In achromatopsia for certain colors it is occasionally noticed that mixed colors—*i.e.*, combinations of these indistinguishable colors—are recognized. This shows that the persons see, but that they are not aware of it, or that they do not want to see.

Bilateral hysteric amaurosis is a very rare phenomenon. The pupillary reaction to light is intact, but may be decreased and, through spasm of the sphincter pupillæ, may be absent for the time being. In some cases (Mauthner, Knies) the pupils were said to have been rigid and dilated (?). This condition, which tends to follow a spasm, lasts only a short time,—a few hours or days.

I observed a case of this kind, in which the blindness lasted for months, and at one time for over three years, and which recurred thirteen times within a decade. The eyeballs were constantly in a position of convergence, and could only be brought from this position slowly and partially, the upper eyelids hanging far down as in partial ptosis (without there being any orbicular spasm present); the pupils were narrow, but reacted to light. In another case (Haslau) the amaurosis was said to have lasted for a year.

It has been claimed that those affected with hysteric amaurosis orient themselves better to their surroundings than do other blind persons, that they get out of the way of obstacles, etc. In the case described above I was unable to satisfy myself of the truth of this fact.

Unilateral hysteric deafness does not, on the whole, impair hearing very much. One rarely observes the patient inclining his sound ear, in order to catch the sound with it. He is, indeed, often unconscious of any disturbance; the opinion has even been expressed that unilateral deafness is never noticed in binauricular hearing. Bilateral deafness is generally a transient phenomenon. Osseous conduction is almost always lost under these conditions, and Rinne's test is negative. An hysteric deaf and dumb condition has been observed several times (Ballet, Mendel, Francotte). Subjective noises in the ear often accompany hysteric deafness. The sensory disturbances of hysteria are often accompanied by cutaneous and mucous anesthesia of the corresponding entrances of the organs of special sense (external auricular canal, etc.).

Those troubled with hemianesthesia and other sensory disturbances have frequently no suspicion of this defective sensation. They notice that something is wrong, complain, for instance, often of pains in a part of the affected side,—*e.g.*, of unilateral headache, which radiates towards the arm and shoulder, but it is only during the physician's examination that they become aware of their anesthesia. Hyperesthesia may be combined with the anesthesia; a region on which painful disturbances are painless may be very sensitive to the slightest touch.

Hysteric disturbances of sensation are very *variable*. They may come suddenly and leave as quickly,—after a spasm or a sudden emotion or a slight injury. In some cases, however, they are more constant.

It is an interesting fact that the anesthesia may be influenced in many cases by manipulation, either displacing it or removing it. Hemianesthesia is especially susceptible. It may be made to disappear by the use of a faradic brush. The anesthesia may be shifted to the other side (*transfert*) by placing a metal or magnet upon the insensitive skin, or *vice versa*. The disorders of the special senses change at the same time. The opinion expressed that the same metal does not act on every person has not been proven, and less reliance can be placed upon the view that the metal given inwardly produces a cure. This shifting may also be accomplished by the use of sinapisms, wood bones, true and false magnets, etc. It is probably purely a psychic influence, a form of suggestion. Some authors have been inclined to ascribe a specific influence to the magnet, but sometimes the anesthesia is not transferred but is made to disappear, after having been repeatedly shifted from one side to another. At times this transfer occurs very quickly, in other cases it is only noticed after a half-hour or longer.

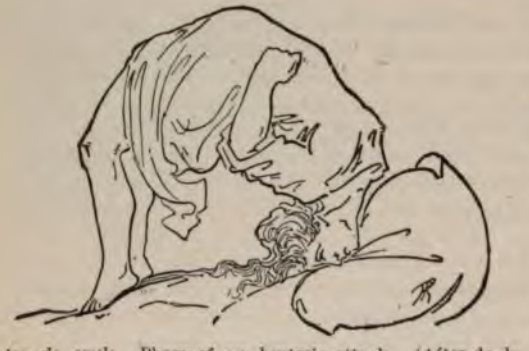
Disorders of Motility; Symptoms of Irritation.—Spasms belong to the symptoms of hysteria, and are absent in very few cases. There are many forms. Especially typical are the *emotional and respiratory spasms*. Crying and laughing may be so severe as to become spasmodic. The respiratory muscles also become involved. The intensity of the emotion displayed, its duration, and the inability of the individual to prevent it are the characteristic signs. The respiratory muscles also may be affected independently, a quickening of the respiration with a feeling of fear and anguish occurring paroxysmally, is a frequent form of these spasms. In one case every noise heard produced it. In these conditions of *tachypnea* no objective signs of want of air are observed. Clonic twitchings of the *abdominal muscles* of chronic duration are likewise sometimes noticeable. *Singultus*,[1] *eructations, spasms of yawning or sneezing* (a continued repetition of these respiratory phenomena lasting for hours), are sometimes seen. A *spasm of the vocal cords* happens more rarely. This is a paroxysmal spasm of the laryngeal muscles, which narrows the glottis and produces asphyxia,—a condition which has many times made a tracheotomy necessary. This spasm may only occur in attempts at phonation (*aphonia spastica*). *Tussis hysterica* is a severe, raw, dry cough, similar to barking or yelping. Each attack may last some hours. Hysteric coughs always disappear in the night. A common form of spasm is the *blepharoclonus* and the *blepharospasm*, which is often combined with photophobia, watering eyes, etc.

It is not settled whether *globus*—one of the most common symptoms of hysteria—should be classed as a spasm or a paresthesia. The patient has a feeling of pressure in the throat as if a ball was tightly clasped there, or a sensation of a ball passing upward from the stomach to the throat. It is not probable that local spasms of the pharyngeal or esophageal muscles are the cause of this. There is, however, a form of hysteric dysphagia which seems to depend upon a spastic closure of the alimentary tract. All muscles which are under the influence of the will may be the seat of hysteric spasms. A twitching of an extremity, or more than one, is observed, with repeated *rhythmic stereotyped movements*, or a wild, irregular to-and-fro movement of the limb. They are, however, always movements that can be produced voluntarily. But the hysteric patient produces them with a strength and stamina impossible to a normal person, or, if able, only for a short time.

---

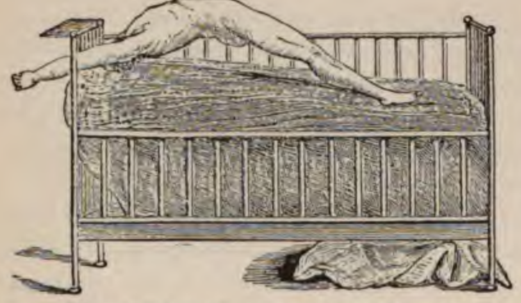
[1] An epidemic of this symptom has been observed several times, and very recently by Abeles.

FIG. 268.



Arc de cercle. Phase of an hysteric attack. (After de la Tourette.)

FIG. 269.



Phase of an hysteric attack. (After Charcot.)

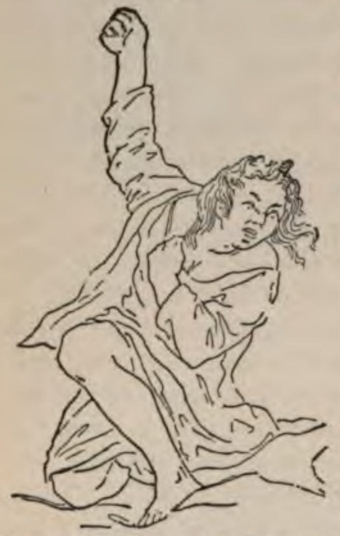
During these localized spasms consciousness is generally not lost, but may, however, be clouded or be accompanied by *hallucinations* or *diseased conceptions*.

To understand the nature of *general hysteric spasms*, a description of the severe types, the attacks of grand hysteria (Charcot) must precede.

In well-developed cases the attack occurs in four distinct phases or periods. We often find a premonitory stage, also, with the following prodromes: Depression, increased irritability, fear, palpitation, globus, etc.; then occurs the *aura*, a sensation of a ball passing from the ovaries or stomach to the throat, combined with marked *fear*, tinnitus aurium, palpitation of the heart, a mist before the eyes, and a clouded sensorium. Next follows the *epileptoid period*, a condition of spasm very similar to that in the first stage of an epileptic attack. The eyes close; the patient falls to the ground (not suddenly, as in epilepsy, and without injuring himself); the head is drawn backward or towards the side; the masticatory muscles are tightly contracted, or, if only partially, the tongue is extended; respiration stops; the face becomes red and congested, and finally cyanotic; the arms are stretched out or adducted upon the trunk; the hand is clenched, and the legs extended at all joints. After this stage of tonic rigidity, which does not last very long, a stage of *clonic spasm* follows, in which the cyanosis disappears; respiration returns, but is more rapid than normal, and very loud, and the pupils, which at first had been contracted, dilate. These spasms inaugurate a new period in the attack, that of *contortions and grand movements* (*clownism*). The patient makes horrible grimaces, alternately flexes and extends the limbs and body, varying constantly from one movement to another; he clenches his hand as if in anger; throws his leg into the air; crosses them; makes salaams with his upper body; inclines his head backward, and produces the so-called *arc de cercle*, an exaggerated opisthotonos, the body being rolled from one side to another, or around its longitudinal axis, thrown against the wall, etc. (Figs. 268 and 269). Crying, bellowing, scolding, yelling, frenzy, laughing or crying spasms, may accompany this muscle-delirium. Next comes the period of *passionate* (plastic) *attitudes*. The expressions of the face and the posture of the body conform to the hallucinations of the patient. Fever, fright, anger or delirium, ecstasy, eroticism, meditation, are represented by the patient in such a manner that an actor could justly be jealous. These hallucinations may often be influenced by external stimulation. Next follows *quiet delirium*, in which animal hallucinations play a prominent part. The paroxysm gradually decreases, and often leaves some disorder in sensation, movement, etc. There may be only one attack noticed, which has a duration of from a quarter- to a half-hour; but in some one attack follows another without any noticeable interval, and often one period may be continued for days, constituting the so-called *status hystericus;* or an *état de mal* may be present, lasting for some days. In this, in contradistinction to the status epilepticus, the temperature remains normal.

Partially developed attacks occur more often than these grand attacks. They consist of one or more phases of the latter; but every possible variation in the grouping of symptoms may occur. The attack may confine itself to the aura and the first epileptoid attack; and epilepsy will be so closely simulated thereby that careful observation is necessary to make a diagnosis. The hysteric attacks are accompanied by more active movements, and betray the influence of the volition. Consciousness is almost never completely lost, and the signs of unconsciousness are therefore absent; above all, the reflex pupillary rigidity. External influences also increase the severity of the attack, the convulsions becoming more severe when the patient is approached, and it is attempted to quiet him. Or the face assumes a characteristic expression; the body describes an *arc de cercle*, or some other peculiarity of hysteria shows the nature of the attack. The tongue is scarcely ever bitten, though the hysteric frequently bites the lips. The prevention of an attack by pressure upon the ovaries is often of diagnostic importance.

FIG. 270.



Phase of an hysteric attack (*attitude passionelle*). (After de la Tourette.)

A combination of hallucinatory delirium with clonic muscular twitchings or respiratory spasms may also represent the attack.

An hysteric seizure may occasionally be similar to the type of *Jacksonian epilepsy*,—*i.e.*, confined to one side of the body, commencing in certain muscles and extending to the others. Consciousness is dulled, but not lost. These attacks may be excited or stilled by psychic suggestion or from the pressure-points. There is no increase in temperature. They may remain confined to one extremity for a long time before becoming general. Attacks similar to *petit mal* also occur in hysteria.

Hypnoid Hysteric Conditions.—A number of hypnotic phenomena which have been considered to be different stages of an hypnotic condition, and which may be produced artificially in hysteric individuals and may pass into each other, may also occur spontaneously in this neurosis. To these belong *catalepsy*, *lethargy* or *hysteric conditions of sleep*, and *somnambulism*.

Catalepsy, which may occur in other *psychoses* also, develops not only suddenly as a result of psychic excitement, but also periodically without any known cause. The most characteristic symptom is the rigidity of the extremities, which, when moved passively, remain for hours or days in the position given to them, and offer as little *resistance* to attempted movements as if they were of wax (*flexibilitas cerea*). The heart and respiratory functions are weakened, and may be slowed. Sensibility is lost, also the reflexes, though the corneal reflex is almost always present.

Consciousness may be intact; at least the patients often say that they could hear, but are unable to answer. They are more often, however, in a kind of dream, and are under the influence of hallucinations.

The eyes are generally closed, the glance is staring, and the face expressionless. Cold douches or faradism sometimes succeed in arousing the patient from this condition, which may last for days, weeks, or months. There are also partial attacks of catalepsy, in which the rigidity does not involve all the limbs. The rigidity is also not always wax-like, but the extremities may offer much resistance to passive movements. Catalepsy is an isolated symptom, or combines itself with hysteric attacks of spasm.

Hysteric conditions of sleep (*lethargy*) are of no slight interest. They may occur independently or be associated with attacks of spasms which precede or alternate with these lethargic attacks. Headache may be present as a prodrome, but the sleep comes on suddenly, and on superficial examination the patient seems to be deeply asleep. The muscles are not always relaxed, and all transitions between slight contraction and severe contractures are found. The masticatory muscles are often contracted. A twitching or tremor of the lids is often noticed. The respiration and heart may be markedly slowed, the former even having been seen to stop for some minutes (hysteric simulation of death). The sensory system is variously affected. Stimulation of hyperesthetic zones may excite movements of repulsion, and sometimes even stop the attack. Movements also occur during an attack which give the impression of being volitional or reflex. The skin reflexes are absent, but the tendon-reflexes are always present. The patient awakes slowly or gradually and with total *amnesia* as regards the attack.

By *narcolepsy* is meant attacks of sleep of short duration and of sudden onset, from which, however, the patient may be awakened by the slightest touch. Narcolepsy occurs also in other diseases, particularly as the equivalent of epileptic attacks and from psychic degeneration; it is, however, often hysteric in nature. Conditions of half-sleep, with vivid day-dreams, may belong to the symptom-complex of hysteria.

Somnambulism.—It may occur in connection with an attack of grand hysteria, and forms to a certain extent a prolonged third stage, or appears isolated under the picture of an hallucinatory delirium. Stimulation of the spinal senses often influences the hallucinations and illusions of the patient. In these conditions he may undertake complicated actions and reveal a marked increase in acuity of all the senses. In an attack all the details of previous attacks are recollected, while in the lucid intervals there is an inability to recall anything that transpired in these somnambulistic states. The whole bearing and nature of the patient generally shows that he is under the influence of delirium, which separates him entirely from the outer world. He appears in his "second nature," which may persist for weeks and months, rarely altered very much externally, but his character and disposition seem to have changed. These are the cases in which we may speak of a *double personality*, of the two fold nature of the psychic individual. We also characterize as somnambulism those conditions in which the patient leaves his bed at night, walks around sleeping, and does things of which he has no remembrance when he awakes. This condition may be hysteric or epileptic, or result simply from a general psychic degeneration, without it corresponding closely to any one type. Charcot holds that this somnambulism is rarely hysteric.

Tremor is a symptom of hysteria which deserves special attention, as so many types may occur in this disease similar to those observed in different organic affections, that errors in diagnosis for this reason are sometimes made.

We often see a *vibrating* tremor of rapid oscillations. This is a result of the general nervousness which so often accompanies hysteria, however, and is not an attribute of hysteria. Another form is characterized by oscillations of average rapidity and large amplitude (five to seven a second). Though this tremor may also occur when the patient is at rest, and be increased by psychic excitement, there are, nevertheless, other cases in which voluntary movements evoke the tremor or considerably increase its intensity. An intention tremor similar to that of multiple sclerosis is, therefore, the result. It, however, is not as dependent upon active movements as in multiple sclerosis, lasts longer, may appear when the patient is at rest, and may be absent during some movements. A form similar to that of paralysis agitans has also been described, though this has been in cases which were complicated and not positively hysteric in character. The tremor is often of an indefinite, variable character (polymorphous).

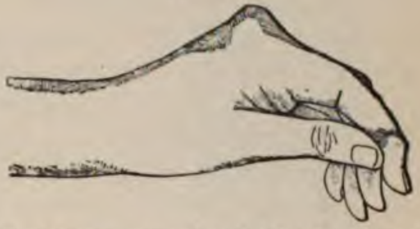
The tremor may be persistent or occur only in paroxysms; it may be slight or so severe as to resemble a shaking palsy. If the legs are particularly involved, the tremor may offer an obstacle to walking. On the other hand, I have several times observed the tremor and shaking become especially severe whenever the patient assumed a recumbent position. Psychic influence, particularly the induction of an hypnotic condition, was always sufficient to quiet it. The tremor may confine itself to one or more extremities or to the leg and arm of one side, or extend over the entire body.

A neuropathic disposition may reveal itself in early childhood by the presence of a tremor. The appropriate chapters must be consulted for the hysteric form of myoclonus and of general tic.

Hysteric contracture—*i.e.*, the fixation of a limb in a certain position by reason of continued muscular contraction—develops spontaneously or as a result of attacks of spasms, or of a trauma, pain, etc., in the paralyzed parts. It often limits itself to one extremity or a part of one, to one side, or in some cases to all four limbs.

The *nature of the contracture*, the *deformity* produced thereby, and the *reaction to psychic* influence are characteristic. In attempts, which are very painful, to overcome them, the muscular contraction increases, often as soon as the limb is merely touched, so that attempts to approach the points of insertion do not conduce towards relaxation, in contradistinction to contractures which accompany true hemiplegia. If the arm is attacked, it is generally adducted at the shoulder-joint, is flexed at the elbow-joint at an acute or right angle, while the hand is strongly flexed, rarely over-extended, and the fingers are clenched or are fixed in a writing position (as in tetany and paralysis agitans). (Figs. 271 and 273.) I have also observed a flexion contracture of the hand and fingers.

FIG. 271.



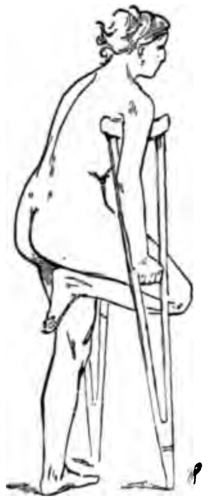
Hysteric contracture of the left hand. (Charcot and Richer.)

The lower extremity is commonly stretched out, the leg rigidly extended in all joints, the foot in marked plantar flexion, and the toes flexed or over-extended. I have seen a flexion contracture (Fig. 272) occur in hysteric paraplegia and paraparesis, or a flexion contracture in one and an extension contraction in the other leg.

An equinovarus position of the foot with contraction of the tibialis anticus muscle also occurs. The contracture may limit itself to a definite muscle-group, as, for instance, to the interossei.

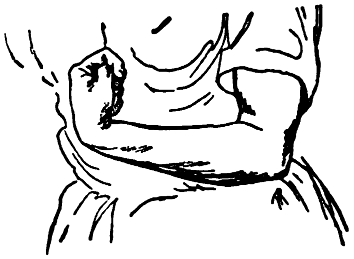
The *joint contractures* are of special interest. They are found in combination with *neuralgia* or hyperesthesia of the joint, and on account of the pain may simulate an arthritic disease. *The knee and, next, the hip* are the most frequent seats of these troubles. They generally commence with pain, which, when the hip-joint is attacked, is found in it and the knee. They generally, however, extend much farther than the joint, radiating to the gluteal, lumbar, and inguinal regions. They do not tend to exacerbate at night. If contracture of the muscles around the joint occurs, the limb may assume a position like that in coxitis, and resemble it so much that even experienced surgeons have recommended operations. The joints and bones are less sensitive and painful than are the soft parts. Even the elevation of a fold of the skin may be more painful than ramming the head of the femur into the acetabulum (Brodie). The sensitiveness is also not limited to the joint, nor is the contracture confined to a few muscles, but generally extends more or less over the entire body. When the attention is distracted, the pain, sensitiveness, and contraction are less. The patient is either unable to walk or he walks peculiarly, with excessive limping. The skin in the neighborhood of the joint is usually unchanged, but may be reddened and somewhat swollen, though never as much as in abscess. The temperature is normal.

FIG. 272.



Hysteric flexion contracture of one leg. (After Richer, taken from Tourette's work.)

FIG. 273.



Hysteric contracture of the left arm. (After Richer, from Tourette's work.)

In doubtful cases chloroform narcosis or hypnosis may be used to establish a diagnosis. In these conditions the muscles relax, and it is easily recognized that the joint is free. Slight nodosities, however, are said sometimes to form on the cartilages.

If the contracture is of long duration, slight atrophy of the muscles may be found, which causes a permanent contracture. The other symptoms of hysteria, however, always support our diagnosis, particularly a homolateral hemianesthesia, as they are rarely absent.

Hysteric contractures may disappear spontaneously and quickly from sudden emotion, cramps, or the influence of psychic therapy. I saw a case of hysteric contracture of the legs, which had existed for over a year, suddenly disappear when the patient in an hallucinatory delirium attempted to throw herself from the window. This contracture may also be voluntarily simulated. To do this, however, great concentration of the attention, strength, and energy are necessary, and Charcot has shown that, as a result of this, irregularity of movement and of the respiration occurs, which is absent in similar hysteric conditions. Hysteric contractures may persist during sleep, but this is not commonly the case.

In many cases a tendency to contracture is present, and can be produced by mere rubbing of the skin, pressure, massage, etc., as also by an active massage (Charcot, Richer).

Contractures are also said to occur in the musculature of the abdomen, as in a case described, in which a contraction of the gastric muscles simulated a stricture and furnished the indication for laparotomy.

Paralysis.—We often find a general or limited weakness or pseudoparesis. The patient apparently strains himself, but the efforts are in vain.

More often, however, it is simply a *loss* of *energy;* the patient does not seem to try to do anything. He clasps the examiner's hand very slightly, and seemingly has no strength to press it. If, however, we endeavor to excite his strength reflexly, by suddenly attempting to pull the patient along by his hand, we can notice a distinct increase in strength. The muscular weakness is also partly due to the fact that volitional impulses are not rightly divided, either in that the action of the antagonists is excessive or that the strength is directed to other muscles. Hysteric paralysis is never confined to one muscle or the muscles innervated by one nerve, but involves the whole limb or part of the body which the patient imagines represents a unit, or the complex of muscles which produce one function (speech, etc.). It is never combined with degenerative atrophy. An atrophy from inactivity may later develop, though occasionally a considerable decrease in volume may happen early (Babinsky). Paralyses in the form of *monoplegia*, *hemiplegia*, or *paraplegia* occur. They commence suddenly. Only conscious voluntary movements are lost, automatic reflex movements being present, at least to some extent.

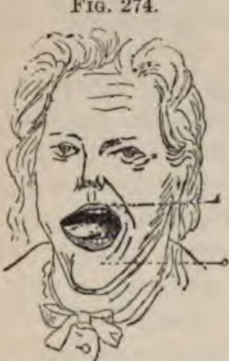
The limb may be used for gesticulation, or is moved in alcoholic and chloroform intoxication. If you grasp a paralyzed hand and manipulate it, you will occasionally experience a slight contraction; especially when the patient is asked to support himself,—*i.e.*, when he is in danger of falling,—he may use his muscles. Terror or anger may also contract them. Often we notice that an arm brought to a certain position by passive motion will remain there a moment, an action which can only occur by use of the otherwise paralyzed muscles.

In the case of a patient who was unable to lift her leg from the bed, I passively flexed the thigh, by supporting it with my hand, and asked the patient to institute movements of resistance in the ankle-joint; when I suddenly drew away the hand supporting the thigh, it remained in the elevated position, the patient having concentrated her entire attention upon the movements of the foot.

A pure *monoplegia*—*i.e.*, a complete paralysis of one extremity with normal movement in the other limb of the same side—is found only in hysteria. The paralysis may be confined also to a hand or a foot.

Hysteric hemiplegia rarely involves the facial and hypoglossal muscles. At times a marked and often an exaggerated deviation of the mouth and tongue, produced by contraction of the muscles of the other side, occurs (hemispasmus glossolabialis). (Fig. 274.)

FIG. 274.



Glossolabial hemispasm. Fibrillary twitching at *b* and *c*. (After Charcot.)

The contracture may also affect the muscles of the hemiplegic side so that a deviation occurs just the opposite to that which is observed in true hemiplegia. That such a spasm is in play may be recognized from the tension of the muscles, particularly on the tongue, in that this organ when forced to the other side returns immediately to its extreme position. The half of the tongue which is affected by spasms appears thicker and smaller. The cheek is not blown out in expiration. Folds around the angle of the mouth and a fibrillary twitching also give evidence of the existence of a contracture. In true facial paralysis a light passed before the mouth is more easily blown out by the paralyzed side. Only in rare cases is a real paresis of the corresponding facial nerve present. Sometimes we are unable to distinguish how much is contracture and how much is paralysis. I observed in one case a *glossolabiomaxillary hemispasm*. Hysteric monoplegia or hemiplegia is either a flaccid paralysis or it is combined with contracture with the characteristics described above.

The *gait* is peculiar and does not correspond to that of true hemiplegia. The leg is simply dragged, not moved in a circle, as in true paralysis, or the patient uses a crutch and does not touch the floor at all. We often notice that the foot is dragged along with the whole plantar surface or the heel on the floor. The gait sometimes reminds one of the manner in which children walk on stilts.

Abasia is not rarely observed,—absolute inability to walk and generally also to stand, with normal movements in a recumbent position. In addition, many forms of hysteric disorder of gait (dysbasia hysterica) happen which imitate almost all types of organic diseases, but all of which have the same peculiarities: exaggerated and forced movements, normal movements when the patient is lying on his back, disappearance of the disturbance under psychic influence or when the attention is distracted, etc. One of my patients could run but could not walk; another could walk backward easier than forward. A common form is a kind of spastic gait in which no rigidity is present; the patient also does not walk upon his toes but upon the whole sole or on his heels. In many cases the gait reminds one of that observed in cerebellar ataxia; the patients perform movements, however, which resemble those of a snake, or which seem to be forced, and which thereby reveal their hysteric nature. In one case of mine, which occurred after influenza, the gait resembled the waddling type of dystrophy. The patient could not hold her trunk erect in sitting, but found it necessary to support it with her hands; the paralysis was localized in the lumbar-pelvic-thigh muscles, but there was no atrophy or pseudohypertrophy, and the influence of suggestion made its hysteric nature evident. A painful contracture of the lumbar and pelvic musculature is more often the disturbance of gait.

The knee-reflexes are never lost, but, if anything, are increased.

Aphonia hysterica is the most frequent form of hysteric paralysis, the voice being in close relationship with the emotional life. It generally commences suddenly, following upon factors which have been mentioned above, particularly after some psychic excitement; it may also be due to reflex action,—for example, to a catarrh of the larynx. The loss of voice is generally absolute, the patient being only able to speak in a whisper. A laryngoscopic examination shows that the vocal cords, in attempts at phonation, either cannot be brought out of their position of abduction or can only be partially adducted. Occasionally a paralysis of the internal thyro-arytenoid is present. The characteristic position produced by paralysis of the transverse arytenoids is observed more rarely. Unilateral paralysis of the vocal cords is probably never seen. Coughing and hawking, notwithstanding the aphonia, are generally accompanied by vocal sounds, as are also expressions of pain. Some patients are even able to sing. Anesthesia or hypesthesia of the pharynx and laryngeal entrance is generally present. Hoarseness is more rare than hysteric aphonia, and is mostly accompanied by a piping voice.

The aphonia tends to disappear as rapidly as it came, spontaneously (from some emotional excitement) or from some psychic excitement or under the influence of treatment. It remits very easily, and in time becomes rather persistent.

Mutism, or hysteric dumbness, is more rare. This condition was studied particularly by Charcot and Cartaz. It is characterized by a complete inability to form tones. The patient exerts himself in vain; no tone, not even a whisper, is produced. The lips and tongue in attempts at speech act as if they were completely paralyzed, although they may be otherwise moved, or are distinctly contracted during speech, without any sound being heard. It is characteristic that the patient understands everything and is nevertheless more mute than an aphasic who can generally say a few syllables or words. His mimic actions (in contradistinction to aphasics) are very pronounced. Phonation may also be intact and the mutism be only partial: the patient by strong efforts is able to pronounce the first syllable of a word and then after a pause a second one, but can go no further. This differs also from that observed in aphasia, where the patient can say nothing or constantly reiterates certain syllables and words. This form marks the transition to *hysteric stuttering,* which generally is a result of (or precedes) mutism. A patient with mutism can read and write. I observed only one exception to this rule,—a mute hysteric patient of mine who was also unable to write. Charcot has also described a case of this kind. The attack, which is generally evoked by some fright,—in two of my cases the mutism developed in individuals who had been close by when lightning struck a dwelling; in another the sight of a mouse brought it on,—lasts for hours, days, or weeks, and may persist for more than a year. We have spoken before of the combination of mutism with hysteric deafness.

Hysteric stuttering, which may also occur independently of mutism, resembles the ordinary form, or the word is torn apart, as it were, in a rather irregular manner: the patient repeats the same syllable or letter several times, and after a short or long interval emits the succeeding ones, a word being spoken rapidly or explosively in between. The muscular action accompanying it seems to be very much forced. Disturbances of respiration may be absent or, on the other hand, may persist when the patient is at rest. The same is true of the twitchings of the facial muscles. Remak in one case thought he could refer the stuttering to an intention tremor, which was noticeable upon movement in other parts of the body also. The absence of intermissions is also regarded as being characteristic of the hysteric nature of this speech disturbance.

A speech disturbance analogous to the syllable stumbling of paretics has been occasionally observed in hysteria.

Hysteric *paralysis of the palatine velum and of the deglutitory muscles* is very rare, and should be diagnosed only when satisfactory reasons for the assumption of hysteria are present and when every complication can be excluded. The velum paralysis causes a nasal voice. The dysphagia is generally an absolute one. I treated a patient who had to be fed for months with a sound. If her nourishment were forcibly placed in the mouth it would immediately flow out again. Dysphagia may also be present with normal articulation. Lost palatal and pharyngeal reflexes are frequently met with in hysteria.

The muscles of the eyeball are rarely paralyzed. A ptosis can occur which almost always proves to be a *pseudoptosis,* due to spasm of the orbicularis palpebrarum. The muscular contraction is felt on attempts to open the lid, and a contraction or fibrillary tremor of the muscles of the lid is observed. If the patient throws his head back the ptosis disappears. The eyebrows also are lower than upon the normal side, just the opposite condition to that observed in true ptosis. A paralytic ptosis is much less frequently met with. It cannot be easily diagnosed from a true paralytic ptosis, as has been observed by Hitzig and myself.

In some cases a spasm of the levator palpebræ superioris is observed.

The most frequent form of hysteric ocular disturbance is either *monocular diplopia or polyopia.* When one eye is closed, double or multiple vision occurs in the other. This diplopia is probably due to defective accommodation, especially to a spasm of the muscle of accommodation (see page 80). It may be combined with *micropsia* and *macropsia.*

Diplopia may also be due to contracture of the extrinsic ocular muscles.

We do not know much that is reliable concerning hysteric *paralyses of the ocular muscles.* They are generally simulated by contracture of the antagonists; for instance, spasm of the internal rectus may simulate paralysis of the externus. A forcible contraction of the internal recti may resemble the picture of external ophthalmoplegia. On the other hand, associated lateral movements do not result voluntarily, but only automatically, as a result of habit, etc. According to my experience, a true hysteric paralysis of the ocular muscles never occurs. The observations of Wilbrand and Saenger, Roeder, Donath, Nonne and Beselin, Hitzig, A. Westphal, Karplus, and Cramer seem to indicate, however, that an apparent paralysis of the ocular muscles may occur in hysteria. This seems to be the case for myosis, paralysis of the muscle of accommodation, and pupillary rigidity (in the widest sense of the word). Even here a spasm of the antagonists may simulate a condition of paralysis. Spasm of the sphincter pupillæ may, for example, appear to be a case of pupillary rigidity. In this respect a communication of A. Westphal is very interesting. He shows that a pupillary rigidity combined with myosis in an hysteric individual occurred only when he concentrated his mind upon it, the pupils reacting and dilating when the examination was undertaken without the patient's knowledge.

A. Westphal reports an interesting observation in a patient in whom he observed a pupillary rigidity in hysteric attacks which afterwards became epileptic.

It is advisable to be very sceptical concerning the occurrence of true paralyses of the ocular muscles in hysteria. *Continued reflex pupillary rigidity especially is not to be regarded as an hysteric phenomenon,* even though it be an accompanying symptom. Always regard it as a complication or simulation (especially secret use of atropine). A common phenomenon is an *insufficiency of the internal rectus* (Landolt). True *nystagmus* does not happen in hysteria. A kind of rapid oscillatory nystagmus, however, occurs (Sabrazés), which can be influenced by reflex action or psychic suggestion.

I have observed the following phenomenon several times: In closing one eye the other assumes a position of convergence, convulsively twitching at the same time, while the pupil strongly contracts.

An hysteric *paralysis of the diaphragm* is rare. Its sudden onset following upon some emotional excitement, the marked respiratory difficulty, observable when the patient is at rest also, the excessive retraction of the abdomen during inspiration, and the other hysteric factors described above render a diagnosis easily established. It should be remembered that one may simulate the symptoms of diaphragmatic paralysis, particularly the inspiratory retraction. Wernicke regards insufficiency of the diaphragm as a frequent symptom in hysteria and neurasthenia. It is an important factor in nervous asthma.

Incontinence of urine is rare; *retention of urine* and *dysuria* occur more often, being due to a spasm of the sphincter. Catheterization is then difficult and *painful*. Pollakuria (strangury with frequent evacuation of small quantities of urine) is occasionally observed. Urinary troubles belonging to the field of compulsive ideas and phobias may occur in hysteria, but will be described at another place.

Gastric Disorders.—Loss of appetite occurs often, sometimes alternating with bulimia. Occasionally all food is refused (*anorexia hysterica*). This condition may become dangerous if not attended to in time. Hysteric vomiting is more frequent; the food is vomited soon after its ingestion; mucus and saliva are also thrown out. The appetite, however, remains good, as a rule, and it is noticed that the patient does not emaciate. Urine has also been noticed in the vomit. This disturbance may persist for a long time, but seldom entails any danger. Morning vomiting may also be a symptom of hysteria.

Obstipation is a rather common symptom, and diarrhea may ensue after some emotional excitement.

In many cases the marked bulging of the abdomen is one of the chief troubles complained of. It is due to accumulation of gas in the abdomen (meteorism, tympanites). It may recede without any gas being evacuated. A noticeable intestinal noise (gurgling) has also been described.

Anomalies of Secretion.—Drooling of saliva, profuse perspiration, unilateral hyperidrosis, may be present, but are not peculiar to hysteria. A transient polyuria may occur, or a diabetes insipidus may accompany the hysteria.

Particularly noticeable in some cases is *oliguria;* it may increase to anuria; that uremia does not come on is due to the fact that vicarious urination occurs, it being found in the vomited matter (Charcot). The physician is, however, often deceived by the patient. In a few cases galactorrhea has been observed.

Vasomotor and Circulatory Disorders.—A rapid change in color—a sudden pallor and flushing—is frequently seen in hysteric patients. *Cyanosis* occurs in the paralyzed and contractured members. A swelling simulating edema likewise may happen; the fingers, however, leaving no pitting on pressure. It is generally hard, elastic, and combined with decreased temperature of the skin. This condition has been called blue edema. A true edema is occasionally seen of an acute circumscribed character. It sometimes localizes itself over a joint, is associated with pain on movement, and simulates a joint-disease.

Local *asphyxia*, also *urticaria factitia*, may occur, though these are not exactly hysteric symptoms. It is often seen in cases with hysteric anesthesia that deep pin-pricks do not cause any bleeding. I excised a piece of muscle in one case of this kind without any bleeding. This phenomenon, which in hemianesthesia does not limit itself to the anesthetic side, is due to a vascular spasm.

Spontaneous hemorrhage on the skin of the forehead, breast, foot, or other locations, is not always a product of deceit. Bleeding from the internal organs is uncommon. It is most frequent from the stomach or uterus. It may take the place of menstruation or occur in conjunction with it. It may be very profuse, and even then, remarkable to say, exerts but a slight influence upon the constitution (observations of Huchard, Charcot, Senator, and others).

Acceleration of the pulse, which occurs paroxysmally and is combined with a feeling of palpitation of the heart, is very frequent, though, notwithstanding the feeling of cardiac palpitation, the pulse may be normal. During the spasm it is normal or accelerated; in the conditions of syncope it is generally retarded.

Trophic disturbances play but an unimportant part. Falling out or sudden graying of the hair has, however, been occasionally noticed. Degenerative muscular atrophy does not occur. The simple atrophy (with quantitative decrease of excitability) which may be present only rarely becomes pronounced. The trophic disturbances which occasionally occur in the skin—even gangrene has been described—are probably almost always (or always?) a result of self-injury or of complications.

The *sexual sphere* is often affected in sympathy. Impotence and decreased sexual power, as well as aberrations of sexual desire (perversions), may be found in hysteria. Loss of desire may be due to anesthesia of the vaginal mucous membrane, but does not always result from this cause. The mucous membranes may also be hyperesthetic, and be the seat of hysterogenic zones. Menstrual molimina are frequently complained of by hysterics, and the other symptoms tend to exacerbate at the menstrual period.

Metabolism is generally not altered. Only during the attacks, according to Gilles de la Tourette and Cathélineau, is it said to be altered, in that the fixed contents of the urine in the twenty-four hours following upon an attack—the urates, phosphates, etc.—are lessened by one-third, and the relationship between the earthy and alkaline phosphates, which is normally one to three, becomes one to two or even one to one. These results, however, need to be confirmed, and their diagnostic importance has been questioned by the authors themselves in that an analogous relationship is observed in other diseases.

We have but few observations of *hysteric fever*. It is, to say the least, very rare, and caution in diagnosing it cannot be too strictly enjoined. Do not forget the possibility of combinations,—tuberculosis, genital diseases, etc. In the cases observed a continued or an intermittent fever was present, the latter being characterized by sudden jumps in the temperature, to 108° F. or 113° F.,(?) by the absence of the corresponding accessory symptoms (alterations of pulse, respiration, and of urine), or by an incongruence between these and the increased temperature, and also by the slight influence exerted by antipyretics. It has also been shown that the daily curve is very large, and that different results are obtained on different parts of the body.

Grouping of the Symptoms; Development and Course.—The variability in the symptoms astonishes all who examine a great number of hysteric patients. There are mild cases in whom only slight objective symptoms are present; others in whom only a single symptom reveals the disease (monosymptomatic), and some in which almost the entire host of hysteric symptoms tend to occur in varied succession. In Germany, and also in the United States, the milder types are the most frequent; women who complain of great unrest, irritability, globus, fear, headache, and other troubles, and in whom a diagnosis must be made from their subjective symptoms, from information concerning previous symptoms of spasm, or from the detection of a sensory disturbance or some other objective symptom. Every transition may be found from these forms to the severe ones, which are characterized by the "grand attacks." In different cases we find some one symptom which appears prominently and tends to establish a diagnosis, and which may even be the only symptom present. It may be an hysteric aphonia, an anorexia, a tympanites, vomiting, pains in many cases, in others paralysis, contracture, or spasms. The monosymptomatic forms (the contractures, paralyses, abasia, hallucinatory delirium) occur particularly in children. The fact that these phenomena are particularly accessible to treatment also makes infantile hysteria characteristic.

If only local symptoms are present and at the time of examination all signs of constitutional disturbance are lacking, we speak of local hysteria.

The disease develops gradually, subjective symptoms introducing it, until a particularly severe symptom attracts attention, or it comes on acutely, marked symptoms coming on immediately after some emotional excitement.

The course is rarely acute. Excluding the rare cases of acute lethal hysteria, there remain still a number of cases in whom hysteric symptoms occur, to disappear in a few weeks, without ever reappearing.

In most cases, however, the course is a chronic one, extends over many years, and may even exist till old age comes on, to disappear with the climacteric or at the beginning of senile involution. I have seen it persist in some cases into the eighth decade of life.

This chronic course is, however, not a stabile one: the condition is subject to great variations, and the clinical picture varies from time to time as in no other disease. The disappearance of severe symptoms and the onset of new ones are particularly characteristic of hysteria. *Long intermissions* often occur in which we do not find any or observe only slight symptoms.

Pathologic Anatomy.—We are not justified in speaking of an anatomic basis to hysteria. The alterations observed in some patients who died were *congenital anomalies of development* or the result of the disturbances in nutrition (inanition). The course, the variability and instability of the symptoms, their dependence upon psychic influences, their sudden disappearance and changeability speak against an anatomic basis in the general sense of the word.

The view that *molecular* alterations in the central nervous system and, particularly, in the brain cortex are the cause of the hysteric phenomena, would best explain the symptoms. Naturally, an exact proof of this assumption cannot be given. There must be an increase of the minute differences in the organization of the central nervous system, which must be accepted as existing in normal individuals also, to explain the differences in the excitability of the various individuals, sexes, and races. At the same time we do not exclude the possibility of these minute alterations extending to the entire nervous system.

It should also be remembered that organic diseases of the nervous system (as, for instance, gliosis) may coexist with the hysteria upon the basis of a congenital anomaly of development, and which should not be wrongly blamed for the hysteria.

Differential Diagnosis.—Generally easily made, a diagnosis may at times offer difficulties. It should always be established by *exclusion.* This is the more necessary from the fact that hysteria is so often combined with other diseases, particularly with organic diseases of the nervous system.

After a careful examination of the internal organs, symptoms must be sought after which are found only in organic nervous diseases. These are ophthalmoscopic evidence of disease of the optic nerve,[1] chronic isolated reflex pupillary rigidity, paralysis of single nerves, degenerative atrophy, Westphal's sign, true ataxia, etc. There are other symptoms which, if they do occur in hysteria, are found so rarely that their presence makes a diagnosis of hysteria doubtful. These are hemianopsia, true nystagmus, bulbar speech, a typical scanning speech, a true intention tremor, chronic incontinence of the urine, internal ophthalmoplegia, etc.; a persistent hemianopsia clearly excludes hysteria.

Hysteria not only may have some symptoms which resemble those found in another disease but may simulate its whole symptom-complex.

It may be difficult to distinguish it from *multiple sclerosis* in those cases in which signs of disease of the optic nerve are not present. The hysteric intention tremor, however, is not so closely connected with voluntary movement, is more clearly dependent upon psychic influences, and is more irregular and variable. Scanning speech is almost always a late symptom in multiple sclerosis; in hysteria a form of speech similar to it may be found at every stage. In such cases, it is pronounced from the beginning but is not equable, some words being scanned and others jerked out irregularly and quickly, others again slowly. If paraplegia exists with contracture, it possesses the characteristics described above, but never assumes the type of a true spastic paresis. If clonus occurs, it almost always appears as if the patient himself produced it, so that it soon loses in intensity and becomes irregular. As multiple sclerosis may, however, occur in conjunction with hysteria, it may be necessary to establish which symptoms are hysteric and which are not.

We find one characteristic of hysteric symptoms always present which serves to distinguish them: *their dependence upon and their reaction to psychic influence.* Every symptom must be examined in this light. Endeavor to distract the attention of the patient and observe the effect, or excite his emotions and watch their influence upon the symptoms. Or assure him that pressure upon this or that place will make the symptoms disappear or will bring out others. The hysterogenic zones should also be brought into use, as attacks may often be excited or aborted by means of them. *Hypnosis* is also a good diagnostic aid. A mere command to the patient to close his eyes and go to sleep will often cause the most objective (the tremor, twitching, contracture, etc.) symptoms to disappear. (Concerning Westphal's pseudosclerosis, see page 215.)

---

[1] I consider it certain that those observations of disease of the optic nerve in hysteria were based on errors, or were a result of complications.

Cerebral tumor and cerebral lues are sometimes diagnosed as hysteria, especially when the objective symptoms are not present, particularly the ophthalmoscopic phenomena. The nature of the headache and the psychic condition of the patient should, however, reveal an hysteric condition. Headache in hysteria, however, may be severe, and cause the patient to scream and cry and gesticulate, while headache in tumors, as a rule, renders the patient dull and apathetic, and betrays itself in the expression of the face, though the patient does not lament; and retardation of the pulse is often observed at the height of an attack. The other symptoms of each are generally characteristic. Vomiting occurs with cerebral tumor, while it is only an accessory symptom in hysteric headache when it has the character of hemicrania.

Hysteric paraplegia may be confused with *myelitis*. The bladder disturbance which occasionally accompanies it is scarcely ever an incontinence, but is almost always a retention of urine due to spasm of the sphincter.

A more important diagnostic sign is the fact that the disorder of sensation almost always exceeds the limits which we would expect in a myelitic focus. It is also, as a rule, accompanied by some disorder of the special senses, and upon closer examination the characteristic psychic anomalies of hysteria may be obtained.

The differential diagnosis between hysteria and other diseases of the spinal cord—*e.g.*, gliosis—has been given under their respective headings.

I have seen (as have Brodie, Paget, and others) cases of hysteria which were falsely regarded as being *caries* of the cervical vertebræ, because nuchal rigidity, pain, and sensory and motor disturbances were present. The sensitiveness upon pressure of the vertebræ was not, however, as circumscribed, and was generally found to be a hyperesthesia of the skin; the contraction of the upper cervical muscles was excessive; and, finally, the limits of the sensory disturbance exceeded those observed in a cervical myelitis.

In one case, in which the patient lay for months in bed, suspended by Glisson's apparatus, I was able to remove quickly all the symptoms by psychic influence and to permit the patient to run about in the garden on the same day.

It has been shown in a previous chapter in what respects *hysteric articular diseases* differ from the true ones. Many errors are made in this regard. In one case which I saw, resection of the knee had been decided upon, and only upon the request of a physician who had been taken into consultation as to the operation, was the patient introduced to me. She came to me on two crutches and left me in a quarter of an hour without them.

In another case, on account of an hysteric coxalgia with apparent shortening of the leg, a boot with a high sole was ordered, which the patient, after a few treatments by electricity, was able to throw away.

Hysteric neuralgia may generally be recognized from its relationship with the hysterogenic zones, its etiologic dependence upon psychic excitement, spasms, etc. Hysteric neuralgia rarely confines itself to a definite nerve area; it is, for example, less commonly observed in a few branches of the trigeminal than in the entire half of the body, and often radiates towards the shoulder, arm, and back. Pressure-points similar to those of true neuralgia are not only found, but also hysterogenic zones from which the attack may be evoked or stilled. The dependence of individual attacks upon emotional excitement is also important.

It cannot, however, be too strongly emphasized that it is important to be very careful and thorough in determining the significance of pain, even when it appears in an hysteric individual, and only to style the pain hysteric or psychogenic whenever a careful examination excludes all possibility of an organic disease of the osseous and articular apparatus, a tumor of the nerve or its vicinity, a constitutional disease, etc. The neurologist, who may easily err in diagnosing an organic from a functional nervous disease, must never forget that there are other palpable diseases which may be the cause of the trouble. Diagnostic errors of this kind, of which I also have been guilty, though fortunately very rarely, have caused me to be silent concerning the errors of others which I had an opportunity of observing.

Hysteric angina pectoris may resemble the true form; the pain generally comes on quickly, is severe from the beginning, and not only radiates into the left arm (particularly the ulnar region) but sometimes over the entire left side or into both arms. The skin may be hyperesthetic, the pulse normal or accelerated, though it has been observed to intermit. Fear and a feeling of syncope are frequent and mental confusion is an occasional symptom. The relative youthfulness, the detection of its psychogenic origin, and the influence of hysterogenic zones are important points.

Hysteric clavus has been confounded with acute meningitis in cases where it was combined with vomiting, opisthotonos, and a slightly retarded pulse. The rapid, apyretic course,—a slight rise in temperature is very rarely observed,—the detection of hysterogenic zones and other hysteric symptoms, together with the absence of all signs of paralysis of the cranial nerves, should guard against such an error.

The chapter on epilepsy should be consulted for a differentiation between *hysteria* and *epilepsy*.

It is not thought to be advisable to give at this place the differential points between hysteria and strychnine poisoning, though they have been confused with each other.

Spasms very similar to those of *tetany* have been observed by Schlesinger, Blazicek, myself, and others, but the characteristic phenomena of this disease (Trousseau's, Erb's, and Chvostek's symptoms), and particularly the increased electric excitability, are absent. The so-called hysteric *chorea* is generally of a rhythmic character. True chorea may be combined with hysteria, or the latter disease may evoke a remission very similar to the former, after the chorea has disappeared, and which may not easily be recognized as being hysteric. *Chorea magna* (so called) is identical with the severe spasms of hysteria, and has nothing in common with chorea.

The term *hystero-epilepsy* was not favored by Charcot, inasmuch as a mixture of the spasms or a transitional condition does not exist. What was formerly considered to be hystero-epilepsy is hysteria; an epilepsy may, however, occur conjointly with and independently of the hysteria. According to my experience, there are persons in whom attacks of an hysteric nature of an epileptic type occur, nor can the occurrence of spasms which seem to be between those of an hysteric and those of an epileptic nature be denied. They were present in individuals who had the signs of a psychopathic diathesis, and did not strictly correspond either to the epileptic or to the hysteric type.

The differentiation of hysteria from neurasthenia is given in the chapter on the latter disease.

Hysteric vomiting need rarely be confused with that due to stomach diseases, and only then when anorexia is joined to it. A characteristic sign is that it follows so closely upon the ingestion of food that the latter is still intact. Toxic diseases (arsenic, etc.) are easily differentiated, inasmuch as pains do not, as a rule, accompany hysteric vomiting, and in doubtful cases the vomit can be examined.

It is very similar to the vomiting of pregnancy, but a gravid uterus can be diagnosed by the anamnesis or by examination. The gastro-intestinal symptoms of hysteria have given rise to many wrong diagnoses,—gastric ulcer, perforation, appendicitis, peritonitis, etc.

Great caution and the widest experience are not always sufficient to prevent a wrong diagnosis, and there is no one, even the most experienced physician, who may not be deceived by hysteria. It is, however, always better to overlook it than to diagnose it where a grave disease is present.

I desire to refer to one other fact: There are persons with a congenital (perhaps due to a developmental inhibition) or early developed weakness in a certain part of the central nervous system,—*e.g.*, the cerebellum, the medulla oblongata, or certain circumscribed areas (centres). In these it may happen that at some time of their life this part fails or becomes so exhausted that it cannot fulfil its functions for some time. Disorders then come on which are not due to an organic disease, and yet are not hysteric in nature, as they do not reveal any dependence upon the mental acts. The disturbances in function may then come on repeatedly in stages.

In a case described by Siemerling, which deviated in many respects from the clinical picture of hysteria, congenital anomalies of development—for example, a cleft in the optic nerve—were found.

Prognosis.—The prognosis as to life is good. Only from spasm of the larnyx or anorexia is death likely to occur, but this is very rare. A few die from suicide. In two cases persistent vomiting was said to have caused death.

The idea that has been recently advanced, that an acute edema may develop in the brain as in other parts of an hysteric patient, is not supported by proof.

The so-called *acute lethal hysteria* is a rare disease. It resembles an acute delirium, with frenzied excitement, spasms, and fever, and ends in death within some days or weeks. Until recently no anatomic changes were found upon autopsy. Alzheimer and Pophoff have discovered, by the newer methods of cell-staining, alterations in the nerve-cells of the brain, whose significance, however, has not as yet been ascertained. Although single symptoms may be relieved, the disease itself can rarely be eradicated.

There is no symptom, no matter how long it has existed, that may not disappear suddenly, either spontaneously or through psychic influence. The prospects for eradication are the better the more recent the disease, the less it has been treated, the more faith the patient has, and the more he desires to be free of his trouble. Hysteric symptoms occurring in children are generally very easily cured, as is also the disease itself. The general spasms and rhythmic muscular twitchings are often very stubborn. Contractures are the harder to cure the longer they have existed (Charcot). To cure the disease itself it is necessary to combat the original cause. As its source is so often due to the position in life of the patient, family, social, pecuniary, or other troubles, change of surroundings and of habits will often effect a cure. I have seen severe cases of hysteria which were cured by the happy marriage of the patient, removing her from unhappy surroundings, while, on the other hand, unhappy marriages have often proven the source of the hysteria. In other cases the disappearance of former causes of worry, the realization of early formed hopes for wealth, etc., have evoked recovery. The persistence of these conditions, however, serves to make the prognosis worse.

The *degree of the hereditary taint* is also of moment. If it is pronounced, if it is revealed at any early age by stigmata of degeneration and marked mental defects, the prospects for a complete recovery are slight. If hysteric symptoms appear in one in whom they were unexpected and whose disposition was at variance with their occurrence, the chances for recovery are much more favorable. Hysteric conditions due to simulation are easily susceptible to treatment.

There is also a class of patients in whom the anesthesia is complete and is combined with complete abulia and finally with generalized paralysis, so that the patient becomes bedridden. Anorexia also occurs, and, lastly, hallucinatory paranoia. In many of such cases death resulted from inanition and complicating diseases. It is still undecided whether these cases should be considered to be hysteric or not.

Hysteria in the male is very stubborn, especially when it is not a pure form, but is combined with neurasthenia, hypochondria, and the different psychoses.

Treatment. — Much can be done by a proper *prophylaxis*. The physician should caution an hysteric mother not to complain in the presence of her children, nor to speak of her sickness, and not to exercise too much sympathy in every little trouble of the child. If this is not possible, the children should be separated from the family. This, of course, will only be decided upon when the temperament or nature of the child reveals signs of hysteria. These children are, unfortunately, the most tender; they are constantly around their mother evincing excessive love, and separation is hard on both. It is then advisable to recommend treatment away from home.

The child should be brought up rigidly and upon set lines, but not harshly or rigorously. Threats are as injurious as is constant ignoring of faults. Every tendency to sentimentality should be repressed. The child should be energetically taught to bridle its passions. To do this requires some will-power on the patient's part, and he must be constantly on his guard. Everything that excites his emotions must be kept away. Popular lectures, theatres, and concerts are on this account injurious. The child should be made to exercise a great deal in the open air;

gymnastics, swimming, horseback and bicycle riding, etc., are of benefit, provided they are moderately indulged in. Neuropathically tainted individuals should be shown the advantage of outdoor work (technical and geographical studies, agriculture, etc.), and their altruistic interests should be awakened; mental strain must be avoided.

If the disease has appeared, the child *should be removed from the family circle,* either to a hospital or into the family and care of a teacher, minister, etc. This is the only way that is successful in many cases. I have seen hysteric boys with hallucinatory delirium cured when removed to other surroundings.

The first duty of the physician is to seek the cause and endeavor to remove it. It may be due to physical conditions, and make necessary the use of iron and a strengthening diet. The pallor of nervous individuals is not always a sign of anemia, but may be due to vascular spasm, and may not be influenced by iron, but by general or psychic treatment. The iron springs of Pyrmont, Elster, St. Moritz, etc., do much good, however, in this class of patients.

The *diet* must be a strengthening one. It should be simple and nourishing, preference being given to milk. If milk is not borne well, kephir or koumyss may be tried, or milk with bicarbonate of sodium or lime-water or seltzer. Spirituous liquors should be interdicted, or limited to a small quantity. Coffee and tea are not so harmful as some think; excessive quantities should, however, be avoided.

When *diseases of the genital apparatus* are present, it is sometimes very difficult to say what should be done. If a gynecologic treatment is advised and is likely to be protracted and the result uncertain, it should not be carried out. If, however, the trouble is slight and can be cured by a single operation, it may be recommended. Chloroform narcosis and the inhalation of laughing gas are not innocuous in hysteric individuals. Necessity is a factor which should be taken into account. Castration has rarely a good influence, never in chronic cases. Naturally it should be undertaken only when the genital disease itself indicates it. Amenorrhea is often a *symptom of hysteria,* as are also often hemorrhages.

In the hysteria of adults, also, a change of surroundings may be the best remedy. It often removes the source of the psychic excitement which has unfavorably influenced the patient. A sojourn in the country, by the sea, or in the mountains may be very efficacious for this reason. Sea-baths, however, are often not well borne by hysteric individuals. Rest in a sanitarium, provided the physician in charge watches the habits of the patient carefully, learns his history thoroughly, and puts himself into his mental life (and is seeking the interests of his patient and not the financial welfare of the institution), is frequently the best measure to pursue. Work is a remedy of much value. It should be adapted to the ability and strength of the individual, and varied as much as possible.

Institutions of manual labor intended for and adapted to the treatment of such cases have lately been established,—for instance, in Zurich.

Some of the *general methods of treatment* are intended to strengthen, excite, and divert the nervous system, and should be prescribed according to the indications laid down in the chapter on neurasthenia. We include here *hydropathic, climatologic, medico-mechanic,* and *electric procedures.* The results are often excellent, but are largely experimental, as their success depends so much upon the trust and faith of the patient.

The basis of the treatment is *psychotherapy.* The physician must betray intense interest in his patient, and must gain his confidence without losing his authority. He should not ignore any symptoms of his patient, least of all,—ridicule or laugh at them,—but should constantly assure him of their curability; must even definitely satisfy him of this. The physician must endeavor to distract the patient's attention from his symptoms, and seek to strengthen his will-power. Much can be accomplished by tact and a proper understanding of the case. He gains in this way the patient's trust, and lays bare the patient's inner life, and shows him the hidden wounds which must be closed before the disease can be cured. Every physician, however, is not suited to every case. In many cases a stern and iron-like bearing is necessary, while every sign of cordiality weakens the trust of the patient.

It is often necessary to combat single symptoms which particularly trouble the patient. Most of our remedies lose their efficacy after a time, and new methods must constantly be used to win the necessary psychic influence. If all remedies of the family practitioner have been exhausted, a little use of authority may be all that is needed to produce a result. A simple "You can," or an ordinary command, may be all that is required in children. In stubborn cases hypnosis may be used. We have no right to oppose an hysteric patient who seeks a cure in holy water or a Kneipp treatment, knowing, as we do, what faith may accomplish in such cases.

The physician must never permit himself to chastise an hysteric person; it is also unlawful.

In addition to psychotherapy and the above-described general treatment, other remedial agents may be used which can reflexly influence the nervous system. These include the *faradic brush,* the *static sparks, cold douches and baths* and other hydrotherapeutic measures, *purgation, massage,* and *gymnastics.*

The measures advised above are applicable to all forms and phenomena. We desire to give a few indications for particular symptoms.

The faradic brush is particularly efficacious against the anesthesia. The application of metals, sinapisms, and magnets may also cure the anesthesia. Coins may be used, or larger pieces of metal, which exert more influence. A large horseshoe magnet should preferably be used. Electric sparks from a static machine act similarly; they often do good when other measures fail.

The *headaches*, backaches, neuralgia, etc., may be combated by the use of the constant current directed to the seat of the trouble. Hydrotherapeutic procedures for the backache, the wearing of an ice-bag, Priessnitz's treatment, etc., are often efficacious. A counter-irritant applied to other places—*e.g.*, the faradic brush to the soles of the feet, or the foot douche—may be very efficacious. The wearing of a galvanic element or a galvanic chain has occasionally acted beneficently. The head-bell or the breeze of a static machine may also be used.

As to *drugs*, one should commence with indifferent and mild remedies: valerian, asafetida, the *bromides* in small doses (five to fifteen grains, several times daily), quinine (two to three grains in powders or pills), etc. If these remedies fail, the *antineuralgics* may be tried. One may strongly influence the patient by administering methylene blue on account of its (to the patient) astonishing action, as the cures reported by Pitres show, though I have had hardly any results from its use.

Narcotics (opium and chloral) can almost always be disregarded. As a suggestive aid, any placebo may be given. I have often evoked sleep by the administration of sugar or gum arabic powders, though all other remedies had failed.

The spasms often resist all methods of treatment. The bromides are much less efficacious than in epilepsy. If a hysterogenic zone be found on the skin from where the spasm may be produced, protect it from all contact; if deep-seated, in the internal organs, a counter-irritant may bring relief (cantharides, cautery).

If an attack has come on, sprinkling with cold water or repeated cold douches may still the symptoms. Occasionally pressure in the ovarian region may abort it; pressure upon the apex region of the heart sometimes achieves this object. Often there is nothing to do but to allow the spasm to die out, protecting the patient from injury and not giving him too much attention. Methodical, intentional neglect (Fürstner) may even accomplish the desired purpose.

Counter-irritation is advisable for the local spasms; if this is not efficacious in cases of spasm of the glottis, an emetic (apomorphine, subcutaneously) may be tried.

In some very severe cases I was able to stop the spasms immediately by suddenly holding a magnet before the patient. Loewenfield recommends hyoscin.

One should institute measures to counteract the contractures as soon as possible in order to prevent permanent disability from muscular atrophy. In addition to psychotherapy, massage is often very efficacious. Tight bandages, especially plaster casts, are always harmful. The use of a constant current, of a magnet, or of the static breeze may be advisable. If these remedies do not succeed the contractures may be cured by evoking an attack of spasm, the contracture often disappearing at its termination.

Aphonia is the most frequent paralytic condition observed in practice. In recent cases the introduction of a laryngeal mirror or of a sound, an indifferent remedy, etc., pressure upon the neck, and similar procedures suffice to entice the voice back. If these measures fail, the faradic current (brush or muscular stimulation) or static electricity may be tried. If this does not succeed, intralaryngeal stimulation of the laryngeal muscles may be efficacious. Cutaneous massage of the larynx, compression of this organ, and a sort of respiratory gymnastics,—have the patient expire strongly, then cough, then produce some sound with a slight cough, and finally a word, etc.,—and also hypnosis may be beneficial. In one case in which every procedure failed, I applied a seton to the back of the neck and caused a permanent recovery.

If the paralysis involves the extremities, it is advisable to request the patient to institute active movements, and at the same time, under continual suggestion, support them with passive ones; the sensations of movement serve to divert the current of innervation into the paralyzed muscles.

In cases of hysteric paraplegia attempts at walking must be made as soon as possible, at first carefully and with the patient well supported, until she learns to move herself, supporting herself upon some stationary apparatus. Recovery is soon an accomplished fact after that. Walking exercises on the horizontal bars have been especially efficacious with me. If there is a tendency for the knees to bend, they should be lightly fastened together. This method is preferable to the "instantaneous recovery" or "agitation-method," in which at one sitting the patient's symptoms are all made to disappear. If all these procedures fail us, we are justified in exciting an attack of spasm, in which in many cases the paralysis relaxes (Charcot). It has been recommended to chloroform the patient, and, before he is completely awake, to set him upon his feet.

It must be remembered that the cessation of a symptom does not mean the cure of the disease, and that a remission is not only possible but probable if it is not possible to remove the basal disease.

Hysteric vomiting resists every treatment. Valerian, bromides, quinine, chloroform (two to five drops in some semi-liquid), etc., may be tried. If not conquered, it is advisable to order a uniform and consistent diet as raw ham, or chopped meat, or to nourish the patient with the esophageal sound or with clysters. In severe cases an emetic may be tried.

Anorexia is a very persistent symptom. Here, also, nourishment through a sound may be efficacious. In many cases in which other remedies failed, a rest cure was beneficial (see chapter on neurasthenia).

Obstipation rarely yields to the ordinary purgatives. The use of laxatives is not beneficial. If slight doses of a remedy do not succeed, larger ones will also generally be inutile. The waters of Marienbad, Homburg, etc., are likewise disappointing. An hysteric patient of mine discovered that the desire for stool, with consequent passage of the same, came on whenever she administered castor oil to her child, while the personal use of this or any other drug was resultless. The drastic remedies are also useless. *Cold-water clysters* are of more advantage, and electric treatment is particularly beneficial in this class of patients—faradization of the abdomen, galvano-faradization with the use of a massage roller, intrarectal electric treatment. Massage may also be tried. The patient should accustom himself to attempts at evacuation at stated intervals.

HYPNOTISM AND HYPNOSIS.

Hypnosis is a condition allied to sleep, but differs from it in the mental bond (*rapport*) which exists between the hypnotiseur and the subject. The former is able to act upon the imagination of the patient, to arouse conceptions in his mind which under certain conditions become his mental property and influence his physical state to a certain extent.

Hysteric persons are particularly, but not exclusively, susceptible to hypnosis. According to some investigators who have made exhaustive studies of this question (Liébault, Bernheim, etc.), about eighty per cent. of all persons are hypnotizable, though the accuracy of this statement has been doubted by many.

Charcot and his pupils who studied the hypnotic condition in women affected with hysteria major believed that they observed three different stadia of the same which led into one another.

1. *Cataleptic State.*—In this the eyes remained open, with a staring expression; the body became statue-like; the limbs could be brought into any position desired, in which they would be kept without any signs of exhaustion. The deep reflexes are said to be absent (?) or decreased. Sensation is lost; the special senses are, however, but slightly impaired.

The cataleptic state may be produced by a sudden sensory stimulation

In some very severe cases I was able to stop the spasms immediately by suddenly holding a magnet before the patient. Loewenfield recommends hyoscin.

One should institute measures to counteract the contractures as soon as possible in order to prevent permanent disability from muscular atrophy. In addition to psychotherapy, massage is often very efficacious. Tight bandages, especially plaster casts, are always harmful. The use of a constant current, of a magnet, or of the static breeze may be advisable. If these remedies do not succeed the contractures may be cured by evoking an attack of spasm, the contracture often disappearing at its termination.

Aphonia is the most frequent paralytic condition observed in practice. In recent cases the introduction of a laryngeal mirror or of a sound, an indifferent remedy, etc., pressure upon the neck, and similar procedures suffice to entice the voice back. If these measures fail, the faradic current (brush or muscular stimulation) or static electricity may be tried. If this does not succeed, intralaryngeal stimulation of the laryngeal muscles may be efficacious. Cutaneous massage of the larynx, compression of this organ, and a sort of respiratory gymnastics,—have the patient expire strongly, then cough, then produce some sound with a slight cough, and finally a word, etc.,—and also hypnosis may be beneficial. In one case in which every procedure failed, I applied a seton to the back of the neck and caused a permanent recovery.

If the paralysis involves the extremities, it is advisable to request the patient to institute active movements, and at the same time, under continual suggestion, support them with passive ones; the sensations of movement serve to divert the current of innervation into the paralyzed muscles.

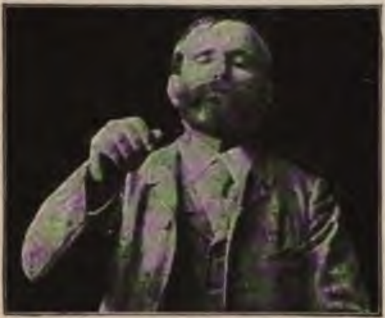
In cases of hysteric paraplegia attempts at walking must be made as soon as possible, at first carefully and with the patient well supported, until she learns to move herself, supporting herself upon some stationary apparatus. Recovery is soon an accomplished fact after that. Walking exercises on the horizontal bars have been especially efficacious with me. If there is a tendency for the knees to bend, they should be lightly fastened together. This method is preferable to the "instantaneous recovery" or "agitation-method," in which at one sitting the patient's symptoms are all made to disappear. If all these procedures fail us, we are justified in exciting an attack of spasm, in which in many cases the paralysis relaxes (Charcot). It has been recommended to chloroform the patient, and, before he is completely awake, to set him upon his feet.

It must be remembered that the cessation of a symptom does not mean the cure of the disease, and that a remission is not only possible but probable if it is not possible to remove the basal disease.

senses slumber, also the will, the memory becomes contracted in scope, and the subconscious state is reached in which suggestibility is increased. The patient should be commanded to sleep, he should be told confidently that "he will soon sleep, that his eyelids are becoming heavy, he cannot open his eyes any longer, his limbs are becoming heavy, that he sleeps, sleeps soundly, still deeper," etc. It is advisable to explain to him that the sleep will not be a complete one, but that the patient will hear all that the physician says.

First attempts may not succeed, or the individual feels only a slight heaviness in his limbs. This indicates that he is susceptible and that succeeding attempts will become more successful until a condition is reached in which his suggestibility is appropriately increased. He then sits with closed eyes, immovable, allows his flaccid arms to be passively raised, where they remain if commanded to do so (Fig. 275); or they fall when all support is withdrawn, etc. That the different motor and sensory functions, the special senses, and the mental state can be influenced in this condition is well known and need not be discussed *in extenso*.

FIG. 275.



A patient under hypnosis.

In this condition all symptoms of hysteria may be made to disappear. It is advisable not to remove them instantly, but to suggest them away gradually, confining yourself to one symptom until it is eradicated before proceeding to another. Hypnotic sleep has also been suggested as a curative agent, allowing it to last for hours and days.

Breuer and Freud have called attention to another use for hypnotism. They found that many symptoms of hysteria were referable to a psychic trauma from which the patient had been unable to unload his mind through an emotional outburst. If it is then possible in hypnosis to bring into consciousness the old trouble and lighten the mind by crying, etc., the evil is removed. The foreign body has been removed from the wound. A simple "awake" or the blowing of one's breath upon the face is sufficient to arouse the subject.

I have deemed it advisable to introduce here the subject of hypnotism, because I believe that it may be used occasionally with good results. It has been particularly efficacious in the symptoms of hysteria, the psychogenous pains, paralyses, anesthesias, spasms, contractures, vomiting, and obstipation. The other methods of treatment must be tried first, combined with psychotherapy, *waking suggestion.* Only when these fail should hypnosis be used.

This treatment seems to me to be also adapted in many cases to combat the different compulsive ideas and conditions of fear. If any one is troubled with concepts which are abnormal or which injure the health of the individual, it is justifiable to direct your treatment directly against these pathologic thoughts and to endeavor to substitute normal ideas for them. This is naturally not meant to apply to nor would it be possible in maniacal conceptions.

It may, however, act injuriously and call into being a severe hysteria, so that great caution is necessary in its use. For this reason, if for no other, only experienced persons should use it, and then only for therapeutic purposes, and after other methods have failed. In doubtful cases hypnosis may be used to establish a diagnosis, as has been shown in the previous chapter.

NEURASTHENIA, OR NERVOUS EXHAUSTION,[1]

is a very common disease to-day, especially in the large cities. Even though it may have occurred at all times (and had been known for a long time as nervousness), it has without doubt increased in extent in the last years by the extra demands that have been made on man in his struggle for existence and in his social life. Some difficulty in deciding upon this point is due to the fact that a close distinction cannot always be made between the slightest grades of this disease and certain phenomena which are still regarded as being physiologic, since normal persons may experience symptoms which, when more strongly marked, are characterized as neurasthenic.

Neurasthenia occurs in both sexes, but in its pure form perhaps more often in males. No age is exempt, but it is essentially a disease of middle life, though children of from five to ten years of age may be attacked, and aged persons may also become neurasthenic.

Heredity is the most important *cause.* A neuropathic predisposition, if present, will tend to produce neurasthenia upon the slightest exciting cause. A *congenital* neurasthenia also occurs, whose origin may be traced to early childhood. We often find physical and psychic degenerative stigmata associated with it. A toxicologic taint (alcoholism of the parents) may also lay the foundation for neurasthenia in the descendants.

Emotional disturbances are an important etiologic factor. A single emotional shock may suffice to produce it. This is particularly true of prolonged psychic excitement. Mental over-exertion must also be considered as an etiologic moment. It is not so much the quiet, equable mental work as it is the irregular, night-robbing, anxious labor which is dangerous to the nervous system. We thus see that speculators, rivals in competitive tests, those preparing for an examination, etc., are frequently the victims of neurasthenia.

---

[1] Beard, Levillein, Bouveret, Loewenfeld, and Binswanger have written exhaustively upon this subject.

Those who work at night, even though they have plenty of time during the day for sleep, very often become neurasthenic. This refers to telegraph operators, night watchmen, compositors, etc. Working in overheated rooms is also a cause. All moments which weaken the organism may act as causes. We often see neurasthenia come on after a severe loss of blood or after chronic fevers, and also after the acute infectious diseases. To what extent this is due to the toxins cannot be decided. Diseases of the genital organs, ear, and nose are not rarely combined with neurasthenia. Individuals with persistent scoliosis are frequent subjects of neurasthenia. That neurasthenia may be of *toxic* origin cannot be denied. Chronic alcoholism, chronic lead and arsenical intoxication, and immoderate use of nicotine may produce neurasthenia.

Syphilitics often become neurasthenic. This is due to weakness of the nervous system produced by the infection and the treatment as well as to emotional excitement. At least the fact that hereditary lues and neurasthenia are often allied indicates this (Binswanger).

The opinion of Bouchard, that this disease is due to autointoxication resulting from disease of the gastro-intestinal tract, does not seem to me to be sufficiently established; and still less Glenard's theory, who wishes to refer it to changes in position of the abdominal organs (enteroptosis, gastroptosis).

Sexual disorders, and especially *masturbation*, may be a cause of neurasthenia. Sexual abstinence has not been proved injurious, as some claim, but could only be in question in neurotic and erotic individuals. Interrupted connection (*congressus interruptus*) is a very important etiologic factor, according to my experience. *Traumata* are also important in the causation of neurasthenia. Cephalic injuries and shocks, particularly when combined with psychic excitement, as in railroad accidents, not rarely entail neurasthenia, though traumatic neurasthenia has a tendency to combine itself with other neuroses.

Symptomatology.—The chief symptom of neurasthenia is the *irritable weakness*,—*i.e.*, the abnormal excitability accompanied by exhaustion, the latter being predominant. The patient is irritable and easily excited; but the excitement, whether pleasurable or otherwise, soon leads to exhaustion, producing and leaving a feeling of weakness and apathy.

Depression is generally present, but is neither deep nor persistent; it results, to a great extent, from reflection over the diseased condition and the hypochondriac thoughts produced by it. A *condition of fear* is almost always present, and is, as a rule, indefinite; the patient feels as if he had done something wrong, broken something, or as if some evil were impending. Often it is a fear of insanity which produces this feeling, sometimes marked enough to cause suicide. The fear may only appear in crossing open places, when mingling in a crowd, on seeing a fire or lightning, hearing thunder, etc. (See the next chapter.) *Inconstancy* and *inability for decision* are often marked enough to be regarded as a symptom. The *intellectual* capacity is never influenced to any extent. The patient often complains that his memory has weakened, that he reasons with difficulty, etc. On close examination, however, the ability to decide, to combine, etc., are not found to be lessened. Concentration upon himself makes it impossible for him to interest himself in anything else, and thus he appears to forget easily. Active *spontaneous mentation* is also reduced. *Fatigue*, however, easily results, his ability for work is markedly abridged, and the least exertion exhausts him. The intensity and duration of this fatigue are characteristic. It may be so marked that all mental work is rendered impossible. Occasionally the ability to conduct visual memory pictures to the brain, to remember the appearance of a certain person, place, or object, is greatly impaired. An artist complained to me that he could no longer judge a picture, because in inspecting one part he would forget the other. At other times the patient will complain of the variations in attention, the haste and instability of his thoughts. Among the important and almost constant *subjective troubles* we must include *headache, vertigo, and sleeplessness.* There are few cases in which these symptoms do not appear at some stage of the disease.

The headache is generally characterized as a head pressure, as if some weight from within were pressing upon the brain, as if the head were splitting, etc. This "*lead-cap*" *headache* is very characteristic. Often it is only a feeling of heaviness and hebetude: "head does not feel free;" "I cannot think freely," etc. My patients would frequently complain that in talking they felt a buzzing in the head, without, as in autophonia, a tubal disease being the cause of it. The character of the headache may be considerably modified by *morbid introspection* and *nosophobia* (fear of sickness). Just as neurasthenia seems to be often combined with *hypochondria*, the single symptoms may also serve as a basis for hypochondriac notions. The headache often leads to the thought that a tumor or syphilis is present. As soon as the attention is directed towards this pain it may increase in intensity, and feel just as, according to the conception of the patient, it would be felt in the sickness that he dreads. The subjective character of this pain, its dependence upon the attention and conceptions can always be recognized.

Vertigo is also a frequent symptom. It is generally a sudden and transient feeling of stupor, a feeling as if the senses had disappeared, as if the patient would fall, or as if the floor was being taken away. True attacks of vertigo with incoördination occasionally occur. The fear of dizziness may influence the patient so much that he does not risk walking alone, and at last will not leave his home unless the energetic insistence of his physician overcomes his fears. A man who for this reason had not left his house for years was able, through psychic influence on my part, to walk and finally to undergo long tours on foot.

Insomnia may sometimes be so persistent as to dominate the clinical picture. Sleep is generally incomplete or restless, or the patient has difficulty in going to sleep, or he awakes too early. The intensity of this symptom is generally exaggerated by the patient. Sudden terror during sleep is often given as a symptom.

Pavor nocturnus of children is often an early symptom of neurasthenia; it may, however, be due to other causes (worms, adenoids, etc.). *Bad dreams* may also be a bothersome symptom. It is a curious fact that many of the symptoms are prone to occur only at night, or at least in greater intensity, so that patients dread the setting of the sun. This applies particularly to the phobias, the urticaria, tinnitus aurium, nervous dyspepsia, pruritus, etc. This is due to the fact that with the absence of the daily worries and noise, the patient concentrates his mind upon his troubles. It may be that the higher centres exercise a regulating influence upon the lesser (sympathetic) ones, and that their influences are weakened in sleep. More often, however, sleep is so superficial that it is disturbed by the ordinary digestive acts and peristalsis.

All the factors are not explained by the above. I treated a neurasthenic who for a number of years had been awakened from sleep by a girdle sensation, which appeared regularly at night during sleep, and never when the patient was awake. In another case severe pains behind the sternum were complained of, which also appeared only at night. The significance of these nyctalgias is not clear.

Disorders of the special senses are also found and likewise bear the marks of increased sensitiveness and exhaustion. The eye and ear are particularly often affected. Seeing stars or spots (*mouches volantes*), a mist before the eyes, fatigue in reading ("the letters seem to swim or run together or dance before the eyes"), increased sensitiveness to noises, buzzing in the ears, ringing, whistling, or murmuring in front of the ears, etc., are frequent and painful and stubborn disorders. Many members of a nervous family who were descended from parents who were closely related were troubled by constantly seeing a green wreath. Sight and hearing may become very painful. In fixing the eyes also, especially in reading, other disorders, as head-nodding, gastric heaviness, a feeling of fear, etc., may come on.

Many of the "asthenopic disorders"—especially the onset of fatigue of the sight—are probably to a great extent due to an increased exhaustion of the muscles of accommodation and of the recti interni. *Sight* and *hearing* are not weakened, and an ophthalmoscopic examination never reveals any disease of the optic nerves. A moderate contraction of the visual field is also occasionally observed in cases of pure neurasthenia. A neurotic impairment of hearing may likewise be combined with it.

Of the *motor* disorders, the common symptoms are *weakness* (not paralysis), *tremor*, and *slight fatigue*.

Although the patient generally complains of weakness, and particularly a fatigue of rapid onset, if the active movements are tested all muscular groups are found intact. Single movements are executed without strength and in a trembling manner, but it can easily be seen that he does not use his whole strength, or does not, or may not, exert himself. The rough strength is, however, not decreased, or only slightly. If asked to press your hand, he does so without exercising much pressure, and only by spurring him on will he exert all his strength. Or the opposite occurs: he puts forth a forcible effort, and then his strength suddenly subsides. This motor weakness never increases to paralysis, is never confined to one nerve area, but is always *general*, and is only occasionally limited to one side of the body or to the lower limbs. It is never combined with atrophy or with alterations in electric excitability.

Tremor is a frequent symptom. This is generally a *rapid vibratory tremor* of small amplitude. Slight degrees of it are found in healthy persons after excesses in smoking, venery, or drinking. This tremor accompanies the active movements, but is also brought on by psychic excitement. A *fibrillary* tremor is also often observed. This tremor affects particularly the orbicularis palpebrarum and oris, the first interosseus, etc., and is apt to occur in psychic excitement or from refrigeratory influences, for instance, in the quadriceps femoris on baring the leg. It may increase to muscular dancing (*myokymia*). In attempts to stand with eyes closed, a tremor of the eyelids is occasionally observed, which does not occur in the same intensity in healthy persons. The so-called essential hereditary tremor, whose forms are variable (Brasch), often reveals a relation to neurasthenia.

The *deep reflexes* are generally increased. This is especially true of the knee-reflex. A slight and inconstant foot clonus is sometimes observed. The reflexes in the arms are likewise often exaggerated. All other signs of muscular rigidity are, however, absent, notwithstanding the statements of Binswanger and others. An increase in the *mechanical muscle* and *nerve excitability* is noticed in many cases. Only in severe emaciation, as, for instance, results from a nervous dyspepsia, can the muscles be so flaccid that the reflexes are decreased, and elicited only by the help of Jendrassik's reinforcement. *Whenever the knee-reflexes are absolutely absent, an organic nervous disease is present.*

No paralysis of cranial nerves occurs. The speech is, as a rule, not affected. In the periods of mental exhaustion, and particularly after sleepless nights, the patient may not be as oratorical as is his wont; he may occasionally find it necessary to search after a word, or may confuse words, displace or drop letters (in speech and also in writing), etc. In an examination confined, however, to a few words no disturbance will be recognized, and, if the patient tries, the most difficult paradigms will be promptly repeated. *Stuttering* is often combined with neurasthenia. A form of mutism of a transient nature is also sometimes observed, especially in nervous children, particularly during school-hours. It is generally due to fear. An insufficiency of the internal recti is occasionally noticed. The *pupils* are generally normal, though a slight difference in size is sometimes seen. This is so rare, however, that a complication with some organic trouble should be suspected. Hippus is not very rare.

Sensory Disorders.—In addition to the headaches described above, *pains* may be experienced in various parts of the body. They are especially frequent in the back, and are often referred to some spinal trouble, but may be found in all parts of the body (extremities, abdomen, etc.). They are rarely very severe, but may be described as such by the patient. If, however, an attack is witnessed, it gives the impression that the patient is not under the control of a severe pain. A *neuralgia* (sciatica, tic douloureux) may be combined with the neurasthenia, but the pains in general are not of a neuralgic character. Only rarely does the patient complain of neuralgic pains over the entire body. *Hemicrania* may also be combined with the neurasthenia.

Paresthesias—formication, a feeling of cold (particularly in the feet) —are rarely missing. They are often of hypochondriac origin. The sensation of going to sleep, often present in certain nerve areas, may also be bothersome. These paresthesias are occasionally noticed in the ulnar region; they may be elicited by slight pressure in every nerve area. They often express themselves by a painful itching, which may lead to a form of *pruritus* and *prurigo*, very persistent, and by producing sleeplessness tend to disturb the general health, etc. *Pruritus ani* is particularly a common symptom of neurasthenia, and, according to my experience, results most often from this cause. I have often observed a combination of *prurigo*, *urticaria*, and *tic-like muscular twitchings* in neurasthenics.

In one family the mother and three daughters were affected with pruritus ani et vulvæ; in all of them the trouble started in the thirtieth year without one knowing about the trouble of the other; another sister had a general alopecia.

Alopecia occurs so often in neurasthenia that its connection with this neurosis cannot be doubted.

The vasomotor disorders deserve special attention, being found in many patients. They complain of a *rush of blood* to the head or of bothersome *flushing*. This may also be detected objectively. The face suddenly (during the examination) becomes covered with diffuse red spots, which may extend to the neck and breast, with increased temperature of the skin. The patient is conscious, as a rule, of this disturbance, and mere thinking of it suffices to bring it out. This *erythrophobia*, or morbid flushing (Bechterew), may be very stubborn and troublesome. Often slight stimulation of the skin may cause an intensive and persistent redness of the same, and occasionally wheals (*urticaria factitia, dermography*). A neurasthenic type of spontaneous urticaria also occurs. With the rush of blood to the head may be combined a pulsation and roaring which is felt over the entire head or at certain places.

The disturbances of the heart are closely allied to these vasomotor disorders. They may be subjective or objective. Palpitation of the heart is an important subjective symptom. The patient feels as if his heart moved faster and louder. This sensation generally corresponds, but not always, to an actually existing exaggeration of the cardiac action. Sometimes the patient feels as if his heart had stopped; this may exist with an actual interrupted beat. If these attacks continue longer, they may be combined with precordial fear, hunger for air, pains in the region of the heart radiating into the arms, paresthesias, a feeling of coldness in the hands and feet, and so forth, and resemble very much an angina pectoris.

Acceleration of the heart's action may also be recognized objectively. Psychic excitement and errors of diet, excessive use of coffee, etc., may greatly increase the frequency of the pulse. *Attacks of tachycardia* also occur, which are plainly entirely independent of psychic influence. They may come on suddenly in the midst of an indifferent conversation. The pulse suddenly becomes small and frequent; may reach a beat of two hundred to a minute, as I have seen it do. It was at the same time small, and at times not palpable. Though this symptom was enough to arouse anxiety, the patient was able to continue conversation; he breathed quietly and ate normally. Though it continued for five or six hours, the general condition was not impaired. Only upon a rapid change of position did the patient experience some vertigo; he could, however, walk a considerable distance. Abnormal introspection was not a factor, as he was in constant good humor, and conversed on matters which took his entire attention. Such attacks are generally short, and combined with cardiac fear and pain. Bouveret speaks of a permanent neurasthenic tachycardia, the existence of which, however, I doubt.

Allorhythmia, arrhythmia, and, more rarely, *bradycardia,* have been observed in cases of neurasthenia and hysteria, especially as complications of a dyspepsia. Irregularity and intermittency of the pulse is, however, more often due to nicotine.

Heart murmurs of neurasthenic origin are rare, though a systolic murmur may occur at the height of an excitement. Drummond claims they are of a *cardiorespiratory* nature,—*i.e.,* only accompanying respiration. Vascular murmurs are also a symptom of neurasthenia. Pulsation of the abdominal aorta has given occasion for a diagnosis of aneurism and projected operations (Hösslin). The pulsation occasionally extended over the whole body, and was very bothersome. The so-called pseudo-aortic insufficiency is probably due to this. The nervous cardiac disturbances appear so prominently in some cases that they are called *neurasthenia cordis.* Gerhardt claims that more than one-half of those who have come or have been sent to him for palpitation of the heart had some functional disorder of this organ, and not valvular disease.

It has been attempted to refer the cardiac and vascular phenomena—the vasomotor and cardiac neurasthenia—to disorders of function of the vagus and sympathetic nerves and to characterize them specially (Gerhardt, Riegel, Lehr, Determann, and others). Lehr particularly has entered into this differentiation. It is hardly possible, however, to analyze closely the symptoms of single attacks and to distinguish a stadium of irritation from one of paralysis.

The sexual apparatus is often the basis of the nervous troubles. The way in which the sexual sense becomes the origin of neurasthenic disorders is generally the following: *Masturbation* was the first link in the chain of symptoms, or—and particularly its onset in early childhood—it was a symptom of a neuropathic disposition. As a result of masturbation, *pollutions*[1] were observed. If these occur often, perhaps three or four times a week, and semen is passed repeatedly in a night, they can directly influence the nervous system and evoke neurasthenic symptoms. Hypochondriasis, however, generally steps in to assist in their evolution. The pollutions worry the patient; he reviles himself for causing them, and is afraid that his spinal cord has or will become diseased; he watches himself and symptoms more and more, and thus in time neurasthenia develops.

It should be remembered that the higher degrees of onanism occur in persons who are congenitally nervous or degenerated (imbecile, or tainted with signs of the insanity of degeneracy). I do not doubt that the tendency itself to masturbate may be inherited. We have then a manifold *circulus vitiosus* (vicious circle): the tendency to masturbate is a symptom of the neuropathic diathesis; and the masturbation produces, on the other hand, innumerable nervous disorders.

---

[1] Krafft-Ebbing avers that excretions similar to pollutions occur also in women.

Spermatorrhea, the passage of semen in the daytime, after urination and, particularly, defecation, without the ejaculation being accompanied by erection and an orgasm, must be separated from pollutions, though they are often combined. It has been thought that in such cases a prostatorrhea existed; closer examination revealed in the secretion the presence of spermatozoa. It is not improbable that this spermatorrhea is due to a paresis of the ejaculatory duct. Rarely do actual ejaculations occur in the day outside of defecation; though it is not uncommon to find that small quantities of semen are passed drop by drop during physical over-exertion. One of my patients claimed that when a student he would pass semen while working difficult mathematical questions. Persistent and continued spermatorrhea is observed only in severe traumatic diseases of the spinal cord, and then but rarely.

Spermatorrhea may also be a result of onanism, but often a *chronic gonorrhea* with inflammation of the prostatic part of the urethra is the cause. This may cause spermatorrhea in any one, but particularly in those who are masturbators or neurasthenic. The circle widens further, in that pollutions and spermatorrhea not alone produce neurasthenia, but are kept up and exaggerated through the latter. *This completes the circuit of sexual neurasthenia.*

Fürbringer speaks of an onanistic neurosis. Krafft-Ebbing attempts to divide the development of sexual neurasthenia into three stadia: (1) the genital local neurosis, (2) the lumbar (spinal) neurosis, (3) the general cerebrospinal neurasthenia. This is, however, an artificial division.

Among the nervous phenomena, *headache and backache, depression, loss of desire to work, confusion, bashfulness, a feeling of physical weakness, and mental vacuity* are the chief ones; to these may be added many others which evoke a fear of spinal disease, and which fear of disease is the characteristic of it. Nervous cardiac symptoms and vasomotor phenomena and nervous dyspepsia are common symptoms of sexual neurasthenia. A very annoying trouble is the persistent *erections*, which occur particularly during sleep. In some of my cases they were the chief cause of the sleeplessness.

A frequent result—and, again, often a cause—of sexual neurasthenia is *impotence*. We do not claim too much when we assert that most of those who complain of impotence are neurasthenics, and generally those who have abnormal seminal losses. It is generally only a decreased ability or a temporary impotence. The desire as well as the ability for erections may also be decreased, or ejaculation follows too rapidly, even before the insertion of the penis in some cases, or occasionally takes too long to occur. Often, perhaps in most cases, psychic moments are in play. Before or during the act, thoughts of failure inhibit and disturb the physiologic occurrences which accompany coitus. The sexual act does not permit of reflection, introspection, fear and doubt, or anxiety. Impotence may in these cases also be the result of sexual excesses, particularly perversions and inversions of sexual acts, and may be either absolute (paralytic) or relative,—that is, absent in natural, present in unnatural sexual congress. These different perversions, which are generally dependent upon psychic degeneration, and which include pederasty, sadism, flagellantism, exhibitionism, etc., will not be discussed here. Severe *bladder disorders* do not occur in neurasthenia. We often find, however, an abnormal irritability of the bladder, in which small quantities cause strangury and even dysuria. Fear and abnormal introspection play a prominent part in producing these disorders also. Fear of being compelled to urinate when in some public place brings on a marked strangury; fear of diabetes acts the same way, causing the patient to urinate many times during a day (pollakuria), without any increase in the quantity passed. This pollakuria may also develop upon a neurasthenic background, in that individuals under certain conditions, particularly in the presence of others, cannot pass a drop (*Harn-stottern*, bégaiement urinaire).

Enuresis nocturna of children also often develops upon a neuropathic diathesis. This is probably a congenital weakness of the sphincter vesicæ which tends later to disappear. A hyperexcitability of the detrusor centre, or an hyperesthesia of the mucous membrane of the bladder may be present.

Digestive Disorders.—These are prominent symptoms. *Nervous dyspepsia* is not an independent disease, but one of the most frequent forms in which neurasthenia expresses itself. We find at the beginning and in the milder cases a feeling of repletion, or a bothersome feeling of pressure in the stomachic region, often accompanied by sour eructations, heartburn, etc. These disturbances are not constant, but occur frequently after meals. The appetite may be normal or absent, but is generally very erratic. A persistent thirst occurs in some cases. In many, painful sensations are experienced when the stomach is empty (gastralgokenosis—after Boas). These troubles are often caused by slight errors in diet, while in other cases they are absent, notwithstanding protracted debauches.

The general health and strength is not impaired in these cases. It is different in the severe forms where vomiting, suffocation, cardiac palpitation, and meteorism occur. Food is gradually refused more and more, partly because the patient fears the results, partly on account of the decreased appetite, until *anorexia* comes on, with emaciation and loss of strength.

Our studies in the *chemistry of the stomach* and its motor functions in these conditions have not reached any definite results. A *hyperacidity* of the gastric juice is often found. The quantity of hydrochloric acid may increase paroxysmally at long intervals and produce attacks of severe pain, resulting in the vomiting of sour masses (gastroxynsis). An acidity has been also observed. Rumination or merycism has been described as a symptom of neurasthenia.

Constipation is generally present; it may be inherited and familial and precede the neurasthenia for years or decades. It may, perhaps, be a cause of neurasthenia. A peristaltic torpor (Kussmaul), or an atony of the intestinal muscles, is generally the cause of the constipation. Increased peristalsis has also been observed in neurasthenics. A tendency to diarrhea is more rare, every emotional excitement causing diarrhea. Mucous masses may also be present in the feces. Neuralgic pains in the abdominal organs are not often complained of. A nervous hepatic colic has been described by Fürbringer. The diagnosis depends upon the absence of icterus, swelling of the liver, and the presence of other neurasthenic phenomena.

Hyperidrosis, general or limited to certain areas, is a frequent symptom; it may become very pronounced and persistent. Patients complain sometimes of *salivation* and often of an abnormal dryness of the mouth. *Polyuria* and *polydipsia* may likewise be neurasthenic symptoms. A temporary *albuminuria* is rarely met with; a *glycosuria* is, however, more common. Albuminuria has been noticed several times as a result of psychic excitement. Oxaluria has likewise been described; large quantities of oxalates being found in the urine, with a lessened desire for urination and occasionally pain. It has been assumed that the oxalate crystals tend to irritate the mucous membrane. I do not agree with those who claim that an excessive excretion of phosphates is characteristic of this disease.

The general *nutrition* need not suffer in the slightest in neurasthenia: many of these patients look the picture of health. The insomnia and the nervous dyspepsia may, however, cause not only emaciation and pallor, but also a pronounced loss of strength. It is more rare to observe a progressive emaciation occur, notwithstanding good nutrition.

There are nervous children who become feverish from the slightest cause. In adult neurasthenics, *fever* as a result of psychic excitement is more rare.

The above description would be an incomplete one if I did not mention the variability of neurasthenia, each case being distinct in itself. Some neurasthenics complain chiefly of this pain or symptom, others of that one, and indeed it may be the only one present. It may be the headache and vertigo, or the insomnia, or the cardiac disturbances, or the sexual disorders, or the digestive trouble, etc. By careful questioning, however, other symptoms will be found which had been distinctly noticed in other epochs of the disorder, but which may now be but slightly pronounced. The division of neurasthenia into cerebral, spinal, motor, psychic, etc., is, however, an artificial one and not practicable.

The following may be included among the objective symptoms of neurasthenia:

1. *Increased tendon reflexes.*
2. *Increase in mechanical muscle-* and more rarely in the *mechanical nerve-excitability.*
3. *Abnormal excitability of the cardiac nervous system, cardiac palpitation,* and the other symptoms of *cardiac neurasthenia.*
4. *Vasomotor and secretory disorders.*
5. *Tremor:* a rapid vibratory and a fibrillary tremor.

Every case, however, does not present all these symptoms, nor should it be expected.

Diagnostic Points. — Neurasthenia must always be diagnosed by exclusion. First satisfy yourself that no organic trouble is present. It should be remembered that in the beginning of phthisis subjective disorders are experienced which have a certain relationship to those of neurasthenia (palpitation of the heart, weakness, depression, irritability, tendency to hyperidrosis, etc.). The same is true of diabetes mellitus. A hidden carcinoma has even been falsely diagnosed as neurasthenia. Arteriosclerosis may give rise to errors. The uric acid diathesis may also induce symptoms which are allied to neurasthenia. Neurasthenia cordis must frequently be differentiated from organic heart lesions. Hypertrophy or noticeable dilatation is never functional; nor are heart-murmurs, which are present at all times, not merely during a paroxysm, and which cannot be classed as accidental. But simple increase of the heart's action, a vibrating apex-beat (F. Müller), increased rapidity, a small pulse, slightly intermittent beats and dicrotism, may all occur in neurasthenia. Though only transient symptoms in neurasthenia, they are generally persistent in heart disease. Acceleration of the pulse in neurasthenia cordis is generally evoked more by emotional outbursts than by physical exertion. Nervous heart affections never cause any compensatory disorders. They are generally combined with vasomotor disorders. Marked pulsation of the small arteries and capillaries may also be of neurasthenic origin. Pronounced acceleration of breathing *without any objective dyspnea* occurs in nervous palpitation of the heart; while in stenocardiac attacks, as a result of organic heart-failure, respiration is retarded and accompanied by inspiratory dyspnea with contraction of the auxiliary muscles. Digitalis produces no results in nervous heart diseases. The general condition of a neurasthenic must also be taken into account, as the symptoms of neurasthenia may point to a nervous cardiac disease, though it must be remembered that organic heart diseases may cause neurasthenia.

Among the diseases of the nervous system from which neurasthenia must be differentiated, dementia paralytica, disseminated sclerosis, cerebral tumor, and cerebral or cerebrospinal lues are the most important. The differentiating points have been discussed in the descriptions of these diseases.

A neuritis or other organic disease is often observed in connection with neurasthenia as a result of a chronic intoxication; especially the metallic poisons. Typical neurasthenia has nothing in common with *melancholia, paranoia,* and the psychoses due to exhaustion, though mixed and intermediary conditions occur, and the borders between them often overlap. *Hypochondriasis,* especially, is frequently found associated with neurasthenia.

The two neuroses, *hysteria and neurasthenia,* cannot always be sharply separated from each other, as both may occur in the same patient at the same time, and many symptoms are common to both. They are, however, by no means identical. Though hysteria is often accompanied by neurasthenic symptoms, neurasthenia may also occur in its pure form. In neurasthenia the *spasms* and *paralysis,* also the *anesthesia* and the *anesthesia of the special senses* of hysteria, are absent. The sudden variations of disposition, the sudden appearance and disappearance of the symptoms do not occur in neurasthenia. The phenomena which tend to appear paroxysmally, as the cardiac palpitation, vertigo, and the like, may also be temporary symptoms in neurasthenia; the basic disease, however, shows a certain constancy in symptoms, although their intensity is subject to oscillations.

The increased reflexes, increased mechanical excitability of muscles and nerves, the rapid vibratory and the fibrillary tremor, and the urticaria factitia are neurasthenic symptoms; but they may occur in hysteria, particularly the first one. In hysteria the chief characteristic is irritability with increased suggestibility; in neurasthenia there is particularly exhaustion and weakness with its attendent lack of energy or desire.

Abnormal introspection is also observable in neurasthenia, produces certain disturbances, and increases those present, but never causes paralysis, anesthesia, or contracture, and does not permit an almost miraculous appearance and disappearance of symptoms. The persistent headache is indeed mostly a symptom of neurasthenia, though there are cases and forms in which there are no other signs of neurasthenia in evidence. Charcot, for instance, desires also to separate adolescent cephalea from neurasthenia.

Nature of the Disease.—We believe with Bouveret and others that neurasthenia is a disease of the nervous system *in toto;* that the alterations of the nervous system which cause it are so minute that our present methods cannot detect them.

Even though Nissl's method has shown us minute alterations in the ganglion cells in conditions of chronic intoxication allied to neurasthenia, the anatomic basis is still in doubt. As far as we know to-day, in accordance with our methods of examination, we cannot speak of neurasthenia as a material disease of the nervous system. The many theories that have been set up in order to enlighten us in regard to the nature of neurasthenia cannot be discussed here.

Course and Prognosis.—It develops slowly in general, rarely suddenly or acutely. It takes a chronic course, as a rule, increases *gradatim* from year to year, or after a certain (short or long) time comes to a stand-still. It generally lasts a few years, its course being broken by remissions and complete intermissions.

The stronger the constitution, the less marked the neuropathic diathesis, and the less advanced the disease is, the better is the *prognosis.* It is more favorable in acute cases than in originally chronic ones, and better in acquired than in hereditary neurasthenia. The association of the latter with the signs of psychic degeneration may particularly cloud the prognosis. As far as the prognosis depends upon the cause, that produced by emotional excitement is most amenable to treatment.

It is often dependent upon the station of life of the patient and his ability to fulfil those conditions which are ordered by the physician, and which are necessary for recovery. Complete recovery is often secured, but the chances for it are less the longer the disease lasts or has lasted. Improvement is possible at every period. Even in incurable cases, the danger of the occurrence of a psychosis is not great. Only when the hypochondriac moment is very pronounced, or a minor psychosis is present, is a termination in a psychosis not exactly uncommon. An organic disease never arises from neurasthenia. When, however, the cardiac nervous system is involved, and there is continual fear of heart disease, the continued emotion causes an incessant acceleration of the heart's action until finally an organic disorder may result. *Atheroma of the heart and blood-vessels*, coming on early in life, has been observed by me many times in such cases. In this way neurasthenia may be the cause of death.[1]

The severe symptoms of neurasthenia may worry the patient into committing suicide. In these, a diseased mental condition must certainly be present, but the border between the psychosis and neurosis is difficult to determine.

Treatment.—The treatment may be successful, but is almost always very difficult. In many respects it is similar to that of hysteria, which see.

Medical advice and caution may do much to prevent this disease, though the prophylactic measures necessary are not under the control of physicians. *Prophylaxis* includes the return to a simpler manner of living, to a former state of civilization, depopulation of our large cities, attacks against the modern hunger for gold, prevention of marriages between blood-relations, degenerates, etc. These and other factors necessary to the hygiene of the nervous system cannot be accomplished, and are, therefore, beyond a physician's aims and objects. The family physician is, however, in a position to work in families for the rational education of the children, particularly insisting in neurotic children upon early counteracting measures against the trouble, and guarding them from all injurious moments. Everything which strengthens and hardens the young body serves as a protection to the nervous system. Everything which overburdens the mind, stimulates the senses, excites the imagination, arouses the emotions, weakens the body, etc., prepares the way for neurasthenia (and hysteria). The indications for education are plain (see page 695). In the *choice of a profession or business* also a certain amount of caution is necessary. The individuality and the nervous predisposition must be taken into account. In general it may be said that every form of manual labor—except those in which contact with some poison is necessary—is suitable, particularly agricultural pursuits. Above everything else, however, should a backward child not be forced to study. The child should be guarded and cautioned against masturbation. We do not know any certain protection against this evil, but careful observation and exclusion of the child from the breeding-places of this trouble (boarding-schools, etc.) may help to counteract it.

---

[1] I emphasized this fact in my first monograph on the traumatic neuroses. Runge observed torsion and dilatation of the temporal artery of the same side in persons who had suffered for a long time with unilateral cephalic congestion. Thoma and his students have referred to similar occurrences in their anatomic studies. Régis, Fraenkel, and others have lately furnished additional data upon this question.

The treatment of the developed disease must depend upon the various causes. If mental overwork is a factor, interruption of all mental activity is imperatively demanded, though do not condemn the patient to complete inactivity, but replace the mental with physical work. The physician should not influence an until then hard working man to neglect his business entirely. I have often seen that from the time that I ordered "complete rest" the nervousness increased considerably, or only then came into view, as the "doing nothing" tended to concentrate the patient's mind upon his body. Work in accordance with his capacity and in proper dosage is, however, a therapeutic remedy of priceless value.

The *causal indication* may render necessary the treatment of some sexual disorder or of the stomach, the removal of worms, etc. It should not be forgotten, however, that the gastric troubles are often the result of the nervousness, and the sexual disorders are often not the cause but a symptom of neurasthenia.

Where possible, it is advisable to free the patient from the web of masturbation which surrounds him. It is dependent upon the physician to judge just how far he can go in describing the evils of masturbation, as some patients, especially if hypochondriac, who see constantly before them the dangers of mental and spinal disease, may be made worse by such descriptions. Popular talks and encyclopedias do a great deal of harm in this manner, more so than can be described. In some cases, however, it is necessary to arouse them to show them the evil of their ways and to spur them on to new life. *Psychic onanism* should also be warned against. A change of surroundings may perhaps be necessary. The best protection against masturbation and its results is *physical exercise*, swimming, boxing, fencing, football, baseball, etc., or severe farming or manual labor from the morning to the evening in the country. Bicycle-riding is also advisable for most neurasthenics.

These are also excellent measures to combat the pollutions and sexual neurasthenia. Mental distraction by means of severe mental work, or the various impressions which a trip into a strange land, a sea-trip, etc., produces, may do good. The local treatment of the urethra with caustics is never advisable; the use of Lallemand's caustic stick and other apparatus is particularly to be warned against. Even when a chronic gonorrhea is present, are these measures often of unfavorable influence upon the general condition; they increase the nervousness, and with it, also, the genital disturbances.

Fürbringer has also come to favor the use of Winternitz's refrigeratory sound (psychrophore), particularly in cases where hyperesthesia of the prostatic part of the urethra is present. This is a catheter-like instrument through which water at a temperature of eight tc fourteen degrees Réaumer (fifty to sixty-four degrees Fahrenheit) is allowed to play into the urethra for about ten minutes. Winternitz himself praises this treatment in chronic gonorrhea, pollutions, and spermatorrhea. Impotence due to these factors is also an indication for this treatment.

The *general treatment* for neurasthenia, to be described later, is, however, the most important.

Neurasthenic impotence may also be often treated with success by the use of the faradic brush—particularly upon the scrotal, perineal, and inguinal regions. Galvanic treatment with the introduction of an electrode into the rectum has been praised. The use of pollution-rings and other "awakeners" is not of much benefit. Painting the penis with collodion, etc., has no permanent effect. Electrotherapy does much good in sexual neurasthenia. For spermatorrhea and pollutions I use the galvanic current: a large cathode on the lower spinal cord, anode of about twenty square centimetres on the scrotal, anal, and inguinal region; stabile, current-strength four to six milliamperes. An intra-urethral faradization may also be of service. Cold douches applied to the lumbar and sacral regions are also recommended. *Lupulin* has been recommended for the pollutions, but with *camphor monobromate* I have done more good. Drugs are not of much benefit, however.

To treat *nocturnal enuresis*, remove the cause, which may be of reflex origin (phimosis, adenoids, worms), give a dry meal at night, wake the patient at a certain time in the night, have the pelvis supported and lying high, and resort to psychotherapy. Use *belladonna*, *atropine*, *hyoscine*, but do not expect much from drugs. Internal faradization, as recommended by Seeligmüller and Köster,—the introduction of a conducting-rod into the urethra for a distance of one or two centimetres, using a painful faradic current,—may be tried. The introduction of an electrode into the rectum may also be found efficacious. The disorder tends to disappear spontaneously, and rarely persists after puberty.

Remedies directed against the impotence—cantharides, strychnine, phosphorus, etc.—are in my estimation of no value. One is, however, often forced to conceal the psychotherapy behind an indifferent drug. Neurasthenics who marry with anxious bashfulness concerning the first attempts at cohabitation, may be carried over the anxious period by the administration of an indifferent remedy, and the simultaneous guaranteeing its efficacy, and sometimes cured for all time. It may be bothersome for the physician to decide whether a neurasthenic with sexual weakness may marry. In my experience, marriage acts beneficially in most cases and helps to reinforce the previously impaired sexual power. If absolute impotence or perversions of sexual desire, or bothersome discharges, are present, the physician should not countenance marriage. It is more than doubtful that Brown-Séquard's testicular serum or spermin is of any benefit.

Constitutional Treatment.—Drugs are of little avail. In many cases they need not be given at all. It is, however, often necessary to prescribe sedatives, and it is then justifiable to administer the different *bromine preparations* (the bromides, quinine hydrobromate, Erlenmeyer's bromine-water, etc.). It should, however, be the rule not to give these preparations for any length of time, perhaps for a few weeks only, or for a longer time when small doses administered at rare intervals have a distinct action. The sensitiveness of nervous individuals is enormous, so that one is compelled to establish the dosage which evokes the desired effect in each case. Though suggestion is a large factor, I do not think that one can ascribe the differences in effect and sensitiveness to this alone. I regard *arsenic* as a drug of much value. In cases of severe and persistent neurasthenia, it should always be tried. If anemia is present, it may be given combined with iron, though the anemia of neurasthenia is often secondary or only apparent, and I am of the opinion that in general too much experimenting is done with iron. Quinine in small doses, strychnine, and combinations of arsenic and strychnine, phosphorus, and zinc may be prescribed as tonics. The other nervines may be used to combat individual symptoms, though one cannot be too careful with the newer drugs, antipyrin, phenacetin, analgen, etc. At any rate, they are not to be used continuously.

The view of Kowalewski, that some cases of neurasthenia are of syphilitic origin, and in which a specific treatment brings about a cure, is not in accordance with my experience.

Psychotherapy is of less importance in neurasthenia than in hysteria, but its benefits must not be undervalued. Without a proper conception of the nature of the disease and of the individual no results of importance are possible; on the other hand, it is astonishing what influence medical advice and encouragement based upon an exact knowledge of the disease, and upon a complete examination of the patient, may have in some cases. There are patients of this kind who need nothing else from the physician except a firm assurance that no severe organic trouble is present. I know a number of neurasthenics who at regular intervals look me up to receive my assurance in this regard.

Hydrotherapy is one of the most important curative factors in this disease. *The cold rubs, sprays, and douches,* lukewarm or cool *half-baths* and *sitz-baths* are particularly efficacious. Cold rubs are borne well by most neurasthenics, if begun at a temperature of 25° C. (77° F.); if, however, the individual sensitiveness is pronounced, partial rubs may still be undertaken with benefit. The treatment can always be varied so as to be suitable to the patient. The sprays and douches, also the pouring of water over a patient, are suitable only for the most resistant. Head douches should never be used. *Wet packs* act as sedatives and often induce sleep. All these treatments may be carried on at home, though, other things being equal, the results are better if done at a well-conducted hydriatic institute under the care of a careful and observant physician. A sojourn in an institution also serves to keep away from the patient many injurious moments which in his own home are harmful. On the other hand, the neurotics brought together in a sanatorium through their constant complaints and conversations are *psychically infected.* The physician should seek to prevent all conversation which refers to sickness or complaints. A command of this nature could, however, not be carried out. The measures advised by Möbius not long ago concerning the foundation of nervous institutions deserve particular attention.

A stay at the sea-shore has generally a favorable influence, though the result cannot be guaranteed beforehand. A cold sea-bath can only be borne by the strongest neurasthenic, and then only in the early stages of the disease. The condition of the heart and blood-vessels must be taken into account in prescribing this change of scene. The baths of the East Sea of Europe are less exciting than those of the North Sea. River baths and cool mud baths have sometimes done good. The indifferent baths of Gastein, Schlangenbad, Johannisbad, Landeck, etc., have been tried with good results in neurasthenia. The iron waters of Pyrmont, Elster, Schwalbach, Cudowa, etc., have also been of service in such cases.

Climatic treatment may do much good. The mere change of scene has often been of benefit. The special indications for the selection of country or forest life, high or low mountains, a stay by the sea, or a sea trip cannot be given here; definite indications and exact observations have also not yet been made. We may, however, say that the high altitudes are not suitable for excitable and very weak neurasthenics, and particularly for individuals who are suffering with a [illegible] oped arteriosclerosis. It must also be remembe [illegible] who need the constant attention of a physicia [illegible] certain air-sanatoria and most likely in instituti [illegible] nervous diseases.

Our resorts are particularly adapted for sum [illegible] it may be advantageous to send patients during [illegible] the South: the Mediterranean coast, Florida, etc. [illegible] land, St. Moritz, etc., may also do good.

Electrotherapy has a wide and not unfruitful field in the treatment of this disease. *Galvanization of the head and back and general faradization, static electricity,* and *electrical baths* are productive of much good in these cases. The results cannot be predicted beforehand; it is a matter of experimentation in every case, which should not be omitted, though it must be done carefully, commencing with weak currents. *General massage, active and passive gymnastics, and medico-mechanical treatment* are advisable for patients with loss of energy and apathy. Zanders's apparatus, the home gymnastic apparatus, stationary bicycles, and rowing machines, etc., can be recommended. As to the *diet,* it should be nourishing, mixed, and easily digestible, never one-sided or excessive. Small meals are preferable to large ones. Individual idiosyncrasies must be observed. *Forced fattening* is dangerous. Alcohol should be sparingly used, if at all. The nonsense indulged in by some who advise cognac drinking is particularly reprehensible. Special symptoms require special treatment. Concerning the treatment of neurasthenic headache, see the chapter on Cephalalgia.

Insomnia demands for a cure regular living, regular and early going to bed without a full stomach, avoidance of reading or mental work before going to rest, and exercise in the open air during the day. If this is not sufficient, a warm full bath of one-half to one hour in length, a hot foot-bath, or a wet pack before going to bed is often of benefit, In other cases general corporeal massage, galvanization of the head, general faradization, and the electrical (faradic) bath will produce sleep. If it is found necessary to give drugs, try bromides.

A change of scene is often sufficient to bring the patient again into the way of sleeping. Wooded mountains are particularly efficacious in this respect. A stay at the sea-side may also be productive of sleep, though this cannot be assured in advance. The previous personal experiences of the patient are of value in deciding these questions. Gastein has often a favorable influence upon sleepless neurasthenics; the incessant roar of its waterfall in some cases produces restlessness at first; they generally, however, accustom themselves to the roaring and then find it calming. This resort has also been found by me to do good in some patients who were suffering from nervous buzzing in the ears (without impairment of hearing). Wet packs over the entire body, Priesnitz's bandages around the abdomen, feet, etc., may produce sleep. The beneficent effect of hydrotherapy in inducing sleep should be universally recognized.

If one is compelled to use drugs, the bromine preparations should be tried first. The hypnotics ought to be eschewed as long as possible. *Paraldehyde,* in doses of three to four grams, and *sulfonal* (one to two grams) are good sleep potions. The latter, however, is not suitable for long use, as it may produce severe symptoms of intoxication, vertigo, ataxia, hematoporphynuria, myelitic symptoms, etc. I regard *trional* as being a good and harmless drug. It is given in doses of from one to two grams. I find that in most cases one and two-tenths grams suffice. This hypnotic also, however, loses in time its efficacy, and remains uninjurious only when it is administered for a short time or at internals. Sulfonal should be given about an hour before bedtime in hot drinks (milk, tea, soup), stirred up well; it is advisable to give an alkali or water at the same time. None of these drugs must be given for any length of time. Little praise can be given to the other drugs inducing sleep. Morphine and chloral hydrate may generally be ignored. Whenever, however, it is deemed advisable to secure a deep sleep for a short time, and to show the patient that it is still possible to make him sleep, nothing can be said against the temporary use of these drugs. One should not refer to the few cases in which chloral hydrate has been given for years without any bad results. Chloralamid (two to three grams) may also be mentioned. We desire to warn against the use of chloralose (one-tenth to three-tenths of a gram).

Nervous dyspepsia often resists the remedial agents used for the treatment of the general condition, particularly the hydrotherapy, electrotherapy, and climatotherapy. In severe cases, in which as a result of the defective assimilation a more or less pronounced emaciation has developed, the treatment recommended by Weir Mitchell and amplified by Playfair often does good. If conducted according to the recommendation of Mitchell, it includes isolation, rest in bed, massage, electricity, and over-feeding.

This treatment must be carried on in a special sanatorium or hospital adapted for it. All visits are to be prohibited, also letters, and the nurse must do everything, not allowing the patient to move, even to feed himself. At first he is given only milk, about one hundred to one hundred and twenty grams every two hours, and increased after three days until in twenty-four hours he is taking two to three quarts a day. After four or eight days some bread and butter may be given, and an egg and then a little meat, until in from ten to fourteen days he takes three full meals and one to two quarts of milk. Malt extract, wine, and beef tea also may be used. One may also order soups from the beginning instead of a pure milk diet, or in addition to it (oatmeal, farina, barley, tapioca, and flour soup).

Somatose has given me good service. As active movements are absent, the conversion and assimilation of his food should be aided by massaging the patient daily for one-half hour, later from one to two hours, and also by daily faradization of his muscles for about forty minutes.

This treatment should last for from six to eight weeks. An abnormal mental condition is a contra-indication, particularly a tendency towards melancholia and paranoia. It may be modified and made milder in many ways; the mild-rest-cure, in which chief reliance is placed upon sufficient rest—in bed or upon a sofa for from three to six hours a day—with forced nourishment, is an excellent measure in cases of neurasthenia in which the strength is insufficient.

Concerning the treatment of the obstipation, consult the previous chapter. In general, the trouble should be combated here also, not by purgatives, but by alterations in the diet and in the manner of living. The use of honey, graham bread, kefir, milk-sugar, apple wine, and fruits (in moderation) may particularly be recommended. Milk-sugar should be used in the milk and other fluids in large quantities,—according to Boas, a teaspoonful three or four times daily. At any rate, one can avoid the drastics entirely, while in some cases it may be advisable to order a mild stimulator of intestinal action. Rhubarb, tamarind, magnesia, cascara sagrada, sulphur preparations, etc., may be recommended.

Nervous palpitation of the heart demands a psychic treatment before everything else. The patient must be made to realize that he has no diseased heart, that his palpitation is of no importance, and that his own observation of it evokes and intensifies it. Mental distraction of any kind during an attack, even a quiet walk or ride, may cut it short. Cold compresses around the heart may often quiet it; the same is true of mustard poultices, etc. Where possible, it is advisable, however, to ignore these measures—also used in organic attacks—and to combat them psychically. Bromide of sodium is the best drug that can be used. In cases of pronounced neurasthenia cordis a general treatment is demanded, and here also hydrotherapy is very efficacious.

The presence of neurasthenia cordis does not even exclude the taking of sea-baths. If, however, there is evidence of cardiac weakness or if the diagnosis is uncertain, one should not advise these strongly stimulating double-edged measures.

Landau has recommended for the troubles of the climacteric the administration of ovarian substance of cows and pigs, which may be obtained in tablet form. Cool baths and compresses, menthol-cocaine ointments, etc., may be used for the pruritus. One of my patients who suffered from chronic pruritus ani felt much relieved after the rubbing in of a tar ointment. The most important factor here also is, however, the general treatment and psychotherapy.

In conclusion, one more word as to the treatment of neurasthenia. *Do not* overtreat your cases. In recent cases, if it is possible to remove the injurious factors at the base of it, it is advisable not to undertake any positive treatment. It does much good to the patient also if he sees and hears that the physician, although he does not doubt the reality of the disease, trusts to nature for a cure. How far one can go in this respect cannot be laid down by rule. I know cases of this kind in which the patient, after he had gone through different treatments in vain, returned to a rational mode of life and recovered. "At last he was healed by the universal balsam of ever-healing nature."

DISEASED CONDITIONS WHICH, AS A RULE, ACCOMPANY NEURASTHENIA OR HYSTERIA, OR A PSYCHOPATHIC DIATHESIS, BUT WHICH OCCASIONALLY MAY BE REGARDED AS AN INDEPENDENT DISEASE.

CONDITIONS OF FEAR (MORBID FEAR).

Fear is a common symptom in the neuroses. It may be an indefinite feeling of anxiety not awakened by any particular cause, or it may be definite concepts and external influences which call the fear into action. The sensation is variously described. It has its seat, as a rule, in the cardiac region, at other times in the head. The patient feels as if his heart were standing still; he thinks that he must fall or that he will "get a stroke." Some explain the condition thus: "It seems to me that I have done something wrong, as if something terrible is going to happen."

The expression of the face often reveals a condition of anxiety, the fear often produces vasomotor, secretory, and motor disturbances; the face reddens or becomes pallid, perspiration breaks out, the saliva ceases to flow, the lips and tongue become dry, a tremor and involuntary movements occur, the pulse and respiration become accelerated. All these symptoms may also be absent. Such obsessions last, as a rule, only a few minutes, but may persist for some time.

In another category of cases, fear arises only after certain external factors come into play, particularly upon the crossing of an open place. This *fear or anxiety of locality*—Westphal's *agoraphobia*—is a very common trouble which develops almost exclusively in those of a neuropathic or psychopathic disposition, and particularly in combination with neurasthenia. A feeling of anxiety comes on in attempting to walk over a lot free of houses or of men; the patient feels as if he could not move, as if he must fall to the ground, as if the space was endless in size, etc. In mild cases, he is able to conquer the feeling and to cross the open space, though under difficulties. On the other hand, the fear, although he realizes its silliness and groundlessness, may so dominate him that he is forced to retrace his steps or to pass the place in the company of some one else or to walk under the protection of houses. As soon as the place has been passed, all fear vanishes.

There are cases in which even the crossing of a ford, the passing a street which has not houses on both sides is sufficient to arouse this fear. Some of these agoraphobiacs feel well only when they are among a crowd; others, on the contrary, become anxious when moving along in a stream of humanity.

A variety of phobias have been described, and their number could be increased at the pleasure of a writer if it were not undesirable to give new names to similar conditions.

We speak, for instance, of a claustrophobia (fear of remaining alone in a narrow space), anthropophobia (fear of being among people), potamophobia (fear of passing across a running stream), astrophobia (fear of storms), pantophobia (fear of everything), phobophobia (fear of fear), etc. Such obsessions may come on in the course of one's calling. I treated a minister, for instance, who was affected with marked fear as soon as he stepped to the pulpit, and on account of which he was compelled to forego his calling for some time.

Only in rare cases does fear constitute the only symptom of disease, so that some authors (Hecker, Freud) wish to regard the "fear neurosis" as an independent form of disease. As a rule, it is accompanied by neurasthenic or hysteric phenomena. Thomsen and I were able to establish the fact that in an attack of agoraphobia a concentric contraction of the visual field existed, which disappeared with the attack.

The view of Freud, that these phenomena are always due to sexual excesses or perversions, does not agree with my observations.

The prognosis of agoraphobia and allied conditions of fear is not very favorable in respect to a return of health; the trouble is generally *persistent*, but tends in time to lessen in intensity, so that the patient learns to combat the fear. Long remissions also occur. Complete and constant recovery occurs in only a small number of cases. I was able to determine in many cases that recovery does occur, for in eliciting symptoms concerning another trouble, I found that years before the individual had been afflicted with agoraphobia.

Some authors include these phobias among the compulsive notions or ideas.

IMPERATIVE IDEAS; COMPULSIVE NOTIONS (WESTPHAL); METAPHYSICAL MANIA (GRIESINGER); MANIA OF DOUBT, FOLIE DU DOUTE (LE GRAND DU SAULLE); NOTIONS OF RESTRAINT.

Conceptions which do not arise from associative action, but which come on without cause, and which forcibly enter into the circle of ideas, so that they cannot be removed, although the individual himself recognizes their alien character and looks upon them as not belonging to the psychic ego, are called imperative ideas.

According to Westphal—others have opposed the idea—they are not due to emotion, but excite secondarily a feeling of fear. It is generally a symptom of the neuroses. Even when it occurs alone it can be seen that a *neuropathic diathesis* is present. Charcot and Magnan, however, go too far in looking upon it as being always a stigma of degeneration, of hereditary insanity.

Their development is favored by conditions of exhaustion and by sleeplessness, or evoked by unpleasant and sudden news. In some of my cases unpleasant news (*e.g.*, the information of the suicide of a friend) has served to bring on the trouble. The idea or thought appears suddenly and unexpectedly, as a rule, appears foreign and peculiar, but the more it is sought to dislodge it, the more energetically it breaks through the circle of ideas and seeks to dominate the scene. The ideas present differ considerably. One will think of throwing himself into the river; another thinks of wounds every time he sees a sharp instrument, etc. These ideas occur also to normal individuals, but can be readily banished, and do not constantly return. They become imperative concepts when they persist and cannot be dislodged. At other times these patients are constantly asking themselves questions concerning God, the world, themselves, etc.,—questions which concern every thinker, but which these patients cannot get rid of, which incessantly occupy their minds and banish all other thoughts. These compulsive ideas become particularly harrowing when they take the form of questions: "What am I doing?" "Why do I do this?" "Shall I dress or undress?" "Why do I dress and not undress?" "Why is my bed in this room?" "Is this my bed?" etc. These senseless questions are laughable to the patient as well as to onlookers, but, though he realizes that they are senseless, he cannot prevent them.

There are other cases in which the patient bothers himself with the seeking after *names*. I treated, for instance, a woman who sought to find the name of every object, and was not satisfied until she had written it down. She had in this way covered sacks of paper-bits with names. Very troublesome is the occurrence of obscene thoughts during prayer. The patient desires to worship, and an obscene word comes to his lips and persists despite all endeavors. The desire may refer to *numbers* also: the patient must count the windows of a house, the steps in a flight of stairs, etc.

Very frequently we find that the compulsive idea—and these to a certain extent are still physiologic—refers to a letter not having been addressed, a cupboard not closed, etc.

A modification of compulsive notions, which approaches those described in the previous chapter as "conditions of fear arising from definite external influences," is the desire for urination appearing on every visit to a theatre or concert or similar places. This trouble generally arises from the fact that the desire arose once under conditions which rendered relief impossible for the time being, and that the remembrance of this situation serves to evoke under similar circumstances the conception, and this produces the desire. This trouble is very frequent among women. The desire for vomiting and vomiting itself may come on under similar conditions. A woman who upon one occasion was compelled to vomit at a social affair experienced vomitophobia, and was compelled to vomit upon every occasion that she ventured in society.

In many, particularly grave, cases it is the fear of dirtying himself that governs the thoughts and acts of the patient. He knows that certain objects are not dirty, but, notwithstanding, the fear of dirt (misophobia) rules him; he avoids touching money, door-latches, and other objects (*delirè du toucher*), or is forced to wash his hands frequently, we may almost say continuously. For example, a woman saw a person run over; soon afterwards she heard that the intestines protruded from an abdominal wound; immediately the idea arose that the dirt was sticking to her and to all objects. She was aware of the silliness of this idea, but for years was unable to free herself from it. Occasionally the idea of seeking the life of another person, particularly the nearest relative, enters the patient's mind and annoys him.

Another modification is that in which ideas and counter-ideas arise compulsorily and involuntarily.

A young lady of sound mind reproached herself because she fancied that every one, and particularly her friends, must wish her harm. As soon, for instance, as she thought of this or that object (dress, pictures, ornaments, etc.), which she knew belonged to some one else, the thought arose, "*you* would like to have that;" to this came the thought, "therefore you wish evil to come to X." She was then forced to construct sentences which to a certain extent served to protect her from these conceptions, as "I do not wish X any harm."

A physician who desired to marry was worried by the fear that he was impotent, but convinced himself of the opposite. One day the thought arose, "the genitals are atrophied." Like lightning this thought shot through his brain. From that time he could not rid himself of the idea, although he knew there was no truth in it. He evolved counter-ideas, to obtund the primary one, as he expressed it, but these also then became compulsive ideas: after "the genitals are atrophied" there followed "the genitals are well developed." And this idea persisted with undiminished intensity.

The preceding description shows that compulsive ideas may become converted into compulsive movements and acts, the execution of which serves to give some relief and quiet. The compulsive acts are usually not executed when they compromise the subject or bring him into conflict with the authorities. Exceptions to this rule are, however, frequent. Another form, on account of which I have been consulted several times during the last few years, is the compulsive desire to run after women, without any true sexual purpose or desire being combined with it. Even feelings of fear and palpitation of the heart may accompany it.

The compulsive ideas may also inhibit normal volition.

Diagnosis.—A confusion with maniacal conceptions must be cautioned against. Compulsive ideas are diametrically opposed to the psychic ego of an individual, he sees their strangeness, and *is aware that they are abnormal.* The ideas of the insane, on the contrary, have become their own property. The patient not only thinks that he is followed, but is convinced of it. It is rare for these hallucinations of the special senses to be combined with compulsive ideas. A person suffering from compulsive ideas is not mentally sick, and must not be regarded or treated as being insane. Such a sufferer is able to perform all kinds of mental labor and learns to control himself so that the trouble is not noticed by any except those to whom he reveals it. In some cases the compulsive ideas force themselves so much to the surface that they completely control his thoughts, extinguish his whole psychic life, and impair his imputableness. It must also be acknowledged that compulsive ideas may also accompany a true psychosis. Middle-aged men and women are affected in equal frequency. It may also occur in younger individuals, and even in children. Indeed, the worst cases of this kind that I have seen have been in children, and it is in just such cases that a recognition of the disease may offer much difficulty. The affection is occasionally combined with general tic or appears in families in which other members are affected with this neurosis.

The prognosis of compulsive ideas is grave. There are harmless forms in which the ideas appear only occasionally, and are not particularly bothersome, for instance, the desire for urination or defecation in a theatre. I also know cases in which the individual became satisfied with the idea, did not lose it entirely, but learned to ignore it so much that his condition was very bearable. In others remissions and exacerbations may occur without the trouble becoming very severe. I have often been told by patients whom I examined for some other nervous disturbance that they had been sufferers from compulsive ideas for a long time. In one lady the phenomena followed a Carlsbad treatment; she was free from them afterwards for ten years, and after a condition of exhaustion they came on anew. A twenty-six-year-old lady, who came to me on account of stuttering and other nervous troubles, told me that from her eleventh to her thirteenth year she was troubled with misophobia. Only rarely has a transition into a psychosis been observed.

It is rare for the compulsive ideas to lead to suicide. The typical forms of misophobia seem to represent the severest form of the disease and to affect the mental state most severely.

The treatment corresponds in general to that of neurasthenia, though the psychic treatment is the chief factor. The definite assurance of the physician that a psychosis is not present, nor need be feared, often in itself does much good. In every method of treatment suggestion must enter. In two severe cases which I observed hypnosis was efficacious. In many others it failed.

It is important to see that the patient has sufficient to do to occupy him. Manual labor (gardening, etc.), also painting, drawing, chess playing, may be recommended. For women, with whom it is particularly difficult to introduce variety into their lives, the making of raised letters for the use of the blind is an occupation which tasks their minds sufficiently. A long journey, in which the patient sees new and pretty things in endless variety, may also be of benefit.

ASTASIA—ABASIA.

This disturbance of function, known for a long time, and studied in recent years particularly by Blocq, Charcot, Pitres, Binswanger, Thyssen, and others, is not important enough to be considered as an independent disease. It is more often a symptom or a symptom-complex arising in *hysteria*, more rarely in other nervous troubles, and is most often due to an *emotional outburst* or to a *trauma*. It is a condition which has also been observed after *infectious diseases* (typhoid, etc.) and CO poisoning.

The condition is about as follows: standing and walking are impaired or entirely impossible without there being detectable in a recumbent position any disturbance of motility, sensibility, co-ordination, etc., or any mechanical hinderance to the movements of the legs. The patient who moves his legs freely in bed, with unimpaired strength and perfect co-ordination, collapses on attempting to stand or to walk, or drags himself wearily along. If walking is only hindered (*dysbasia*) the inability may be due to insufficient exercise of strength on the part of the muscles or to an incoördination coming on in walking or to involuntary movements (choreiform or tremor). In most cases the inability is absolute. The individual may generally move on all-fours. Swimming and other locomotor acts may also be intact. Those affected are sometimes able to walk backward.

Abasia is generally a symptom of *hysteria*, and should be regarded in the same light as aphonia and similar disorders. It seems often as if there were a loss of the memory pictures for the movements of walking.

I have also observed cases which indicate that the disease may belong to the category of *occupation neuroses* (which see), and be analogous to writer's cramp. For just as there the combined action of the muscles for a definite co-ordinated action is disturbed, though they functionate normally for every other action, here, also, the otherwise intact ability for movement is impaired in the execution of movements for walking and standing. The disturbance may be spastic in nature (baso-spasm), may be due to weakness (paralytic form), to tremor, or, finally, depend upon the fact that an abnormal innervation of the muscles in walking and standing evokes pain.

In cases of the latter kind walking is not entirely abolished, but the inability comes on after a few steps have been taken, or only a troublesome restraint in walking seems to be present. Hysteric symptoms may be absent, though two of my patients (as also those suffering from writer's cramp) were neurasthenics. In one case over-exertion of the muscles of the leg had preceded, in another, a painful disorder of the foot.

Bruns speaks of a "stuttering of the legs." It is doubtful whether the cases, in which a feeling of anxiety ensuing upon attempts to walk is the cause of the inhibition (stasobasophobia after Debove, Mingazzini, and others), should be classed here. (See chapter on Agoraphobia.)

It is natural that this condition occurs particularly in youthful individuals and somewhat oftener in women than in men. *Cerebellar ataxia* is also a disorder of gait which fits in the definition given above, but it has such definite characteristics that its differentiation is easy.

The prognosis is favorable, though this disorder may be very persistent. For the treatment, see the chapters on Hysteria and Neurasthenia. Psychotherapy and methodical gymnastics are especially serviceable. In children, simple suggestion suffices to produce a cure.

AKINESIA ALGERA (MOEBIUS).

Moebius has described, under the name of akinesia algera (ἀλγηρος, painful), a disease which he regards as a restriction or loss of motion, on account of the pain produced by such movements, without there being any cause for the pains. It is not an independent disease, but merely a *symptom* or *symptom-complex* which arises in neurasthesia, hypochondria, hysteria, and psychic degeneration. At first only certain and especially forced movements produce pain. After some time every movement becomes painful. The pains last longer than the movements, extend to parts which have not been moved, till finally complete inability to move is present, due to pain, not to paralysis. The patient then upon mere inspection may resemble a paralytic.

The pains belong to the psychic algias, the pain hallucinations (Moebius, Erb), and are not, as Bechterew thinks, of a physical nature. In addition to this phenomenon, which is the most prominent, symptoms of *neurasthenia* and *hypochondriasis* are found. The tendency towards the severe *psychoses* is also very apparent. In a case of mine the attacks of pain were accompanied by marked acceleration of breathing and of the pulse. Passive movements also evoked pain. In another case the phenomena confined themselves to the right side of the body and were combined with hemianesthesia. Milder types have often been observed in the course of the traumatic neuroses.

In an analogous manner other functions may suffer and be even completely inhibited, so that they excite pain. There is, for instance, a *persistent form of ocular disturbance* (dysopsia algera) which is due to the fact that mere use of the eyes, but particularly their fixation, evokes eyeache and headache. In several of my patients the sight of white objects (paper, linen, snow, etc.) produced this pain. Nervous persons have repeatedly characterized their chief trouble as a disagreeable sensation (cranial pressure, itching of the head, even dyspeptic symptoms) occurring upon every attempt at reading. In a case observed by Erb the *hearing* especially awakened pain, so that the patient was incapable of conversing or was compelled to indulge in monologue. The pains also compelled him to assume a horizontal position for the past fourteen years.

In one of my cases, every meal evoked these pains, so that in time complete inanition resulted; in the intervals the patient was perfectly well, the ingestion of food, however, always produced these pains accompanied by a pronounced acceleration of the heart, vasomotor disturbances, polyuria, etc. A disease of the stomach was, of course, not present.

The cases described by Neftel as "atremia" probably belong here. In this condition the patient remains in bed because standing, walking, sitting, etc., are accompanied by most painful sensations,—fear, etc., and disturbances of the general constitution.

The prognosis is bad in all these conditions. They generally occur in heavily tainted individuals in whom the *apraxia algera* is the acme of many years of disease. Recovery is, however, not excluded: I successfully treated one case in which the use of the eyes caused pain, by prescribing blue glasses, the employment of the galvanic current, and the administration of arsenic. In Erb's case also, as in the one described by me, which were particularly severe cases of aphagia algera, recovery was effected, according to the personal communication of the patient.

Consult the chapters immediately preceding for the treatment.

THE TRAUMATIC NEUROSES (ACCIDENT NEUROSES).

Injuries may influence the nervous system in many ways. The phenomena and symptom-complexes evoked by gross lesions of the brain, the spinal cord, and the peripheral nerves have been discussed.

Those abnormal conditions which result from concussion and shock, whether the central nervous system is directly affected or it is conducted to the brain by way of the sensory nerves, are of great interest. Their phenomena are to a great extent identical with those of the neuroses and psychoses, above all those of *hysteria*, *neurasthenia*, and *hypochondriasis*. The symptomatic picture occasionally corresponds entirely to one of these neuroses, but generally consists in a *combination of hysteric and neurasthenic phenomena*, and with these are often combined symptoms and symptom-groups which cannot be classed as hysteric or neurasthenic, but which are also functional in character (*reflex epilepsy*, *epilepsy*, *reflex neuroses*, *localized muscular spasms*, *psychoses*, etc.), so that the symptom-complex is a very varied one. A neuropathic and toxic diathesis (alcoholism, saturnism, etc.) favors the onset of these neuroses.

The knowledge of these conditions is of comparatively recent date. They were, indeed, observed and described by earlier investigators, whose interpretation and conception of them, however, was inadequate in that they assumed as the ground of the phenomena a material disease of the spinal cord (chronic meningomyelitis, Erichsen, Riegler), of the brain, or of the peripheral nervous apparatus. It appears to me that the first two cases of nerve-stretching, described by Billroth and Nussbaum in the years 1869 and 1872, should also have been included in the category of the traumatic neuroses.

Our present conceptions have been extended through the investigations of Walton, Putnam, Thomsen, and myself, and the entire disease elaborated at the same time by Charcot (and his pupils) and myself (as also by Strümpell, Page, and others).

In his polyclinic lectures of 1887 and 1888, Charcot says, "These cases until three years ago were unknown to me as well as to others." It is important to remember this fact in contrast to those authors who hold that investigations in this field have merely repeated what had been known in earlier times.

In the **genesis** of this disease, *psychic shock*—fear or excitement—plays an important *rôle:* there are cases in which it alone is the cause of the trouble. Traumata in which a cerebral shock actually occurs in addition to a psychic trauma are particularly liable to evoke these neuroses, as, for instance, *railroad accidents*. These, indeed, were the means of calling attention to this disease. Every injury, however, even those which only involve a peripheral part (hand, foot, etc.), may have this affection in its train. But it is almost always a trauma which is accompanied by a pronounced shock of the affected part of the body or with a severe psychic shock. A part of the paralytic conditions evoked by strokes of lightning (keraunoneuroses) belongs in the category of traumatic neuroses. We find in these cases the phenomena of a functional neurosis accompanied by signs of a material nervous lesion, as I observed lately again, in a particularly severe case.

The symptoms occasionally come on immediately after the accident, but days, weeks, or months often elapse before their onset.

Pains in the affected part of the body generally constitute the first complaint, and throughout the disease are the chief subjective trouble. In the neuroses following upon railroad accidents, the pain has its seat in the back, sacrum, and often in the occipital region. It evokes a *restraint in active motility*, as the patient endeavors to fix the painful parts and to avoid or to restrict as much as possible all movements which will disturb them.

At the same time other phenomena have appeared which are particularly pronounced if the mechanical shock has directly affected the brain (cranial injuries, railroad accidents), or if the accident has been combined with a severe mental emotion. These affect, especially, the *psychic nature:* there develops an *hypochondriaco-melancholic* depression which often reveals itself in the facial expression and in the general disposition of the patient. He is constantly discussing sad thoughts which refer to his ill-luck, his life, and his "hopeless" position. He is at the same time abnormally irritable and sensitive, cries upon the slightest occasion, and appears effeminate. He generally complains of fear, unrest, and terror, and at times severe attacks of fear and, more rarely, of hallucinatory delirium are found. A marked increase in intelligence is rarely observed, though *weakness of the memory* is often complained of. Constant self-observation and the decreased interest in the outer world causes an apathy which may simulate an intellectual weakness. Progressive dementia occurs very rarely.

In the symptomatology of the neuroses which result from cranial injuries *headache* and *attacks of vertigo* predominate. The vertigo may be so pronounced that the patient becomes pale and falls to the floor. In many cases it occurs every time that the patient bends, and is, on such occasions, combined with pronounced reddening of the face and of the mucous membranes (conjunctiva, etc.), and may even be accompanied by a clouded consciousness. *Complete unconsciousness* may also usher in the attack. It is well known that true epilepsy may result from cranial injuries. Spasms and psychic attacks of an epileptic nature have, however, been observed after railroad accidents which have not produced a cranial trauma (Westphal). Injuries may also, in a reflex manner, evoke a definite and characteristic kind of epileptic attack, the so-called *reflex epilepsy* (compare the chapter on epilepsy). It is not justifiable to identify these with *hysteric conditions of spasm*, which likewise are a result of the injuries described above.

The different forms of spasm also, which are not accompanied by a disturbance of consciousness, particularly the *localized muscular spasms*, play a prominent part in the symptomatology of the traumatic neuroses. *Convulsive tic* is also rather common. Phenomena similar to myoclonus (which see) have also been noticed.

Just as we have again found in the symptoms mentioned above the *elements of emotional disease* and of the *neuroses*, the rest of the symptomatology also reveals a series of phenomena which we have learned to know as the sign-posts of neurasthenia and hysteria. We include among the neurasthenic phenomena the *insomnia*, the *general muscular weakness*, and in the majority of cases a pronounced *increase of the tendon-reflexes.* This is in most cases a general increase, though it occasionally happens that it is confined to the side upon which the other symptoms appear, or upon which they are most marked. Here belong, also, the *increased mechanical excitability of the muscles and nerves*, and a number of phenomena which affect the heart and blood-vessels. We may find, for instance, increase in the frequency of the pulse, and more often an abnormal excitability of the cardiac nervous system, so that slight exertion and mental influences suffice to accelerate the pulse.

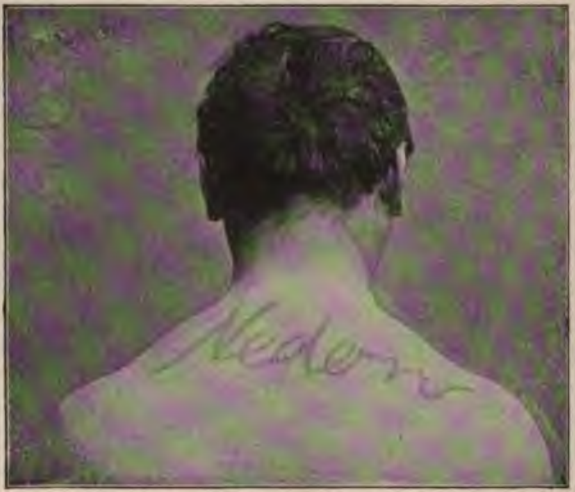
The pulse may also be small; it is sometimes markedly large and soft, similar to a fever pulse, and, according to F. Müller, the sphygmographic curve shows an absence of the elasticity elevation, pronounced and low diastolic elevation with steep and sharp apex (dicrotic pulse).

Arrhythmia of the heart is more rare. A physical examination of the heart reveals nothing abnormal outside of an increased action, though an organic cardiac disturbance may develop from the nervous one (ventricular dilatation and hypertrophy), as I have particularly noticed in many cases of traumatic neuroses. In later stages, an *arteriosclerosis* is frequently observed. Saenger, however, calls attention to the fact that this trouble also occurs otherwise, even in young laborers. In a case of traumatic neurosis of long standing with especial unilateral vasomotor disturbances, I saw an arteriosclerosis develop early in life upon the same side.

Vasomotor disorders are present in the majority of cases: cyanotic discoloration of the skin in circumscribed areas or in larger areas, slight flushing of the face, neck, or breast and neck, and particularly the phenomenon of *urticaria factitia* (dermography). Fig. 276, made from a photograph, shows a man with this phenomenon who had been accused of simulation, who is now insane, and in whom, in addition to other severe symptoms, this phenomenon was very pronounced. The phenomena of Raynaud's disease have also been observed in several cases.

Trophic Disturbances.—Premature graying of the hair and its falling out have been noticed (Stepp, Ladame). I observed an alopecia which began upon the anesthetic side and gradually involved the hair of the entire body. Alopecia unguium has been described by Reid.

FIG. 276.



A case of traumatic neurosis from a fall from a considerable height. The urticaria factitia permits the patient's name written upon his back with a blunt instrument to be distinctly visible. (From a photograph by the author.)

The motor weakness is often accompanied by a *tremor*, which corresponds either to a simple nervous type or to the different forms of hysteric tremor, but may also deviate from these and be similar to that of Basedow's disease, paralysis agitans, etc. It may exacerbate under the influence of excitement and self-observation, or recede if the attention is distracted. In several cases of Nonne and Fürstner (referred to by me before) the chief phenomenon was a slow tremor, which increased during movements, was accompanied by muscular contractions, and impaired both the station and gait. *Fibrillary tremor* is present in many cases, either general in character or confined to the muscles of the paretic side.

It appears in some cases only after over-exertion. Rumpf showed that it may be excited by electric stimulation.

In addition to the general muscular weakness and anenergy of the movements, and the inhibition of motility on account of the pains, *paralytic conditions* may occur, which in most respects differ from those due to organic disease of the central nervous system and which are so similar to those of hysteria that Charcot and others have considered them to be identical with the latter.

The paralysis may localize itself in any part of the body. It may—for example, in railroad accidents, contusions of the spinal region—correspond to the type of a *paraparesis* or *paraplegia*. More often we find a *hemiparesis* or *hemiplegia* in which the facial and hypoglossal are almost always spared. The paralysis often confines itself to one limb. The fact that the unilateral paralysis is always confined to that side upon which the trauma has occurred is of considerable diagnostic value. The functional hemiplegia following cranial injuries corresponds, therefore, to the side of the injury. The character of the paralysis also varies; it is occasionally flaccid, but is more often combined with contracture. And those forms of contracture are observed which also accompany the hysteric paralyses, and which have been described under that heading.

A *pseudocontracture* is occasionally present,—*i.e.*, a portion of the limb corresponding to a contracture without muscular contraction. If the paralysis is incomplete, the effect of movement, notwithstanding the noticeable exertion of the patient, may be slight. This is due to the fact that the impulses for motion are not divided in a correct manner, but reach muscles which have nothing to do with voluntary motion or even from their contraction inhibit them. This may easily seem to be simulated, but can also be pathologic, and is probably due to the fact that the memory for the division of motor impulses necessary to the execution of volitional movements has been lost.

The paralysis is often *absolute:* the affected extremity, upon superficial examination, does not seem to exist for the patient. Sometimes, however, it is possible to see that only the *conscious voluntary* movements are annulled, and that during some emotion or from associative and reflex causes the affected muscles still innervate. One of my patients, for instance, just as he was going to fall to the ground, held himself up by his hand, which was no longer capable of voluntary motion. Another could not move his head by command, but often accompanied the words that he spoke with movements of his head which he unconsciously had formerly used. In the agitated stage of chloroform narcosis, also, the apparently paralyzed limbs are often forcibly moved.

The extremity that has been directly injured is paralyzed the most.

The paralysis is often accompanied by atrophy, which is, as a rule, not marked, and is only accompanied by quantitative disorders of electric excitability (simple decrease).

The disturbances of sensibility and of the special senses are very prominent symptoms. Pains and paresthesias of the most varied kind are almost always present, and objectively detectable sensory disorders are very frequent. An important rule is that these also are found exclusively or for the most part upon the side of the body affected by the trauma. Hyperesthesia is generally found only in circumscribed areas,—for instance, upon the skin of the side of the body that was injured; the anesthesia has generally a larger extension, extending over one side of the body, over arm, shoulder, and breast, over arm and face, or more or less over the entire body. It is often only a *hypesthesia*, rarely a complete anesthesia, and the sensation for pain is particularly apt to be decreased. It should be noticed that with this analgesia or hypalgesia for pin-pricking and the faradic brush, a tactile hyperesthesia of the same part of the skin may be combined. The anesthesia has also, as a rule, the character of that of the special senses (see page 669).

Among the disorders of the special senses, the *concentric contraction of the visual field* has an important place. A perimetric examination is necessary for its detection. It generally involves both eyes, and in cases where hemianesthesia exists it is most marked upon the anesthetic side. It may be the only disturbance of the special senses present, but is by no means a constant symptom.

The anesthesia or hyperesthesia often corresponds to a loss or decrease of the peripheral reflexes, which may be distinctly noticeable when the disorder is a unilateral one. The relationship between the reflexes and sensation is, however, not constant. The different forms of articular neuralgia are particularly *frequent after trauma.*

In many cases the locomotion of the patient is hampered, and *disorders of gait* of the most varied types occur whose character may be difficult to determine. The gait is often impaired on account of the pain and stiffness of the back, as, indeed, the pains of the patient produce the most peculiar positions and postures. On account of the stiff position in which the legs are held, the gait may resemble a spastic one, though the toes do not, as a rule, catch on the ground, nor is there any real muscular rigidity in the recumbent position. A pseudo-ataxic gait develops more rarely; a dragging of the legs in walking, setting down the heels with a stamp without any ataxia being found when the patient assumes a recumbent position, or the gait may resemble that of a drunken person, being combined with a pronounced tremor which increases at every step.

Speech is also often influenced. Mutism is frequently observed after the attack; stuttering, stammering, etc., may also occur. Syllable-stumbling has only occasionally been observed. A paralysis of the vocal cords (adductor paresis) has been observed only in a few cases. The pupils in general react normally to light; pupillary rigidity was observed by me in only a few cases. It is probable that an organic disease complicated these cases, though this symptom also corresponds to the type observed in the neuroses. A difference in the width of the pupils is often observed, which, however, has a pathologic interest only when very pronounced, and when refractive differences are not present. The pupil is generally dilated on the side on which the pains and sensory and motor disturbances are found. The pupillary difference may occasionally be found only during an examination in a dark room or in the shade. *Nystagmus* occurs rarely, though I have several times observed a tremor of the eyeballs which occurred during attempts at movements of them, and which was generally combined with blepharoclonus. In two cases in which the patients complained of headache and vertigo, I observed a transient exophthalmos occur in stooping.

In the few cases of traumatic neuroses in which an *atrophy of the optic nerves* was found, this was a complication, the trauma having produced functional and organic alterations at the same time.

Digestive disorders occur in only a few cases,—anorexia, profuse diarrhea, vomiting. *Nutrition* may suffer a great deal, though it is not rare to find the patient even increasing in weight during the course of his ailment.

Difficulty in evacuating the bladder, obstipation, and impotence are often complained of, though these disturbances can rarely be detected objectively.

Polyuria, *albuminuria*, and *glycosuria* are rare symptoms, though traumatic diabetes may occur with or develop after a traumatic neurosis (Ebstein). In a few cases intercurrent attacks of fever took place. According to recent investigations, the galvanic resistance of the skin of the head is lessened in those cases in which tinnitus aurium, vertigo, etc., occur (Mann).

The differentiation between the *general* and *local* traumatic neuroses as recommended by Strümpell is to a certain extent justifiable. The first show symptoms of a general involvement of the nervous system; in the latter local symptoms appear prominently in the part of the body affected by the trauma. Finally, it should be remembered that the trauma may cause simultaneously an organic nervous disease and a neurosis, so that the symptoms of both may be combined with each other.

Friedmann observed phenomena appearing after head injuries which he grouped under the name of "vasomotor symptom-complex." He included under it headache, vertigo, nausea, intolerance to alcohol, etc.; paralysis of the cranial nerves and fever may also occur. In some of these cases the smaller cerebral vessels were found diseased.

The previous description refers to the *severer* types of the traumatic neuroses. Many cases, however, occur—and they are constantly increasing—in which only subjective troubles exist, complaints of pain, loss of strength, etc., or there is only a slight number of objective symptoms present, or the subjective symptoms do not correspond to any objective phenomena, the examination being negative. These cases are often not nervous patients at all, but are suffering from some hidden surgical disease, as, for instance, Freund showed in a number of cases, and as X-ray examinations frequently demonstrate. A great number of the cases of true simulation belong also in this category.

Pathologic Anatomy.—The name *neurosis* in itself indicates that a pathologic basis to these disease conditions has not yet been found. We assume that in the functional disorders molecular alterations occur in the central nevous system. We can, it is true, refer to only a few autopsies, but these were generally negative. A few observations, however, have shown that shocks which did not produce a direct lesion of the central nervous system caused a disease of the cerebral vessels, especially the capillaries, even arteriosclerosis, hyaline degeneration, and endarteritis obliterans; and it is not improbable that some of the symptoms, as, for instance, the persistent headache, attacks of vertigo, and vasomotor disorders, are due in some cases to these alterations (Kronthal, Friedman, Köppen), though I consider it more probable that these changes are only results of the repeated vasomotor disturbances.

Schmaus has shown that shocks to the spinal cord which are not combined with gross anatomic lesions may produce death of the nerve-fibre or swelling and degeneration of the axis cylinder, medullary disintegration, etc. Bickeles has also shown that blows on the head of an animal can produce a medullary degeneration of the nerve-fibres of the medulla and cord, which are only recognizable by Marchi's method. Such observations show the necessity of caution in one's decisions; they may be particularly adapted to throw a light upon those phenomena which are not psychogenic. Later investigators also (Vibert, Knapp, Crocq, and others) have been inclined to favor the presence of minute material alterations.

Pathogenesis.—According to us the traumatic neuroses are the result of *psychic and physical shock*. Both act mostly upon the cerebrum and evoke molecular alterations in the same areas which govern the higher psychic and the motor and sensory functions and those of the special senses. It is not excluded, however, that finer material lesions (upon the walls of the blood-vessels, degeneration of the medulla of some fibres, etc.) are present, and form the basis of some phenomena. A trauma involving the periphery of the body may also act upon the cerebrum in that the shock is conducted to it by way of the sensory nerves and produces these alterations, or that an irritation arising from a cicatrix continually influences it.[1] Goldscheider has agreed to my ideas in discussing the hemianesthesia appearing immediately after a lightning stroke. Local injuries cause this, particularly when the cerebrum has previously been subjected to an increased sensitiveness (neuropathic disposition) or has been altered before by an accident (shock, emotion).

This theory is opposed to that of Charcot, who regarded traumatic hysteria to be due to *autosuggestion*.

V. Strümpell has lately emphasized the fact that the desire for money, the wish to acquire a competency, the consciousness that he is deserving of it, etc., are a large, if not a chief, factor in the etiology. Although this factor comes in play in so far as the failure of having his desires satisfied and the consciousness of his supposed rights produce depression and irritability, and the desire for money increases self-observation, nevertheless I cannot agree that the symptoms arise in this way.

Diagnosis.—The chief difficulty does not lie in distinguishing the traumatic neuroses from other diseases of the nervous system, but in knowing whether disease or *simulation* is present. As a workman who has figured in an accident knows that damages for injuries may be obtained, it is not uncommon to find these conditions of disease simulated. The occurrence of simulation in cases of the traumatic neuroses was often considerably over-valued, as the nature of these conditions was not understood, and as the examinations of such persons was entered into by those who were without a psychiatric training. The mere fact that in all countries and among the most different people the same clinical picture has been observed to occur after traumata is alone sufficient to prove that we are dealing with real disease conditions. For instance, an Indian physician lately described a case of traumatic neurosis in a Malay, who certainly had no idea of European culture or civilization. Another important fact is that traumata, even where no injury has resulted, may cause these conditions. We must, however, always bear in mind the fact that simulation and aggravation may occur. It is advisable never to undertake an examination with a formed opinion; never to omit to make the examination thorough, and objective symptoms must be earnestly sought for. To these belong continued increase of the knee-reflexes, *mechanical muscle and nerve excitability, fibrillary twitching* or a *tremor* and *clonic twitching* in a few muscles which a normal person cannot cause to tremble alone (triceps, supinator longus, omohyoid, etc.), *atrophy, vasomotor symptoms, symptoms of cardiac neurasthenia, anomalies of secretion*, etc., and *differences in the pupils*. The latter is also occasionally found in healthy persons, but so seldom that it need hardly be referred to.

---

[1] Incidentally I desire to say that slight traumata may also act beneficently upon certain neurasthenic troubles. I know of two cases in which a stubborn, plainly neurasthenic obstipation, present since childhood, disappeared after a fall which caused only a moderate degree of contusion. In another case a hemicrania disappeared after an accident. That traumata, particularly those tending to suppuration, may have a favorable influence upon the psychoses is known and has been lately emphasized again by Bach.

My experience is that a *typical contraction of the visual field* cannot be simulated. Schmidt-Rimpler believes that a campimetric examination is always advisible. In this the visual field must naturally increase in extent the farther away the observations are made, as the visual angle remains the same, while the ignorant simulant does not permit his visual field to increase in accordance with the distance. Such a relationship, in an individual who is not psychically diseased, indicates simulation.

The so-called Foerster's varying type consists in the object used in the test, when introduced in a centripetal direction into the visual field, being seen farther peripherally than when moved in the opposite direction. If, therefore, the object be introduced for the first time into the visual field from the periphery, the second time from the centre towards the periphery, and if in the first test the place where it is visible is noted and in the second the place where it disappears, two visual fields will be secured, the first being larger than the second in every direction. According to the observations of Wilbrand, König, Placzek, and others, this phenomenon occurs in those suffering from the traumatic neuroses more often than in other disorders, and, as Peters also agrees, can hardly be simulated, although others (Schmidt-Rimpler, Peters, Voges) have insisted that this phenomenon is also observed in the healthy, so that this test is not entirely satisfactory. The fact was established by Wilbrand that the so-called expansion in recovery which the visual field experiences in the dark results more slowly under pathologic conditions. The method of measuring the visual fields in the dark, dependent upon this fact, is such a complicated one that it may be omitted in practice.

The observations of Wolffberg, Frankl-Hochwart, and Topolanski indicate a certain relationship between contraction of the visual field and quantitative decrease of the senses of light and color. It is possible that this fact, in further examinations for the determination of nervous visual disorders of traumatic cases, and particularly for the differentiation of disease from simulation, may be valuable.

We cannot enter here into the different methods for the detection of simulation of unilateral or bilateral blindness and deafness, etc. Most of them lose their importance and value in contrast to blindness and deafness of hysteric or psychogenous origin. An apparently valuable method for the determination of simulation of unilateral blindness or deafness has been recently described by Ziehen. In an apparently unilateral amaurosis he places before the patient, one after another, a series of words, which are written upon a slate. With some words he opens the seeing eye, with others it is closed. Afterwards, the individual is asked to give the words he has read, and contradictions will easily occur if simulation has been attempted. The method is suitably modified for the ear. (I abstract this from a verbal lecture of Ziehen.)

Though the constancy of the symptoms is particularly conclusive of their reality, it should not be forgotten that the phenomena of the neuroses are subject to great variability, so that the results of examinations made at different times need not be exactly similar.

Characteristic periodic variations, described by Stern, need to be confirmed by other observers.

Finally, it must be remembered that, though a single symptom or all of them may be simulated, simulation of the whole clinical picture is rarely, if ever, possible.

Von Jaksch has called attention to the fact that the so-called *alimentary glycosuria* belongs to the objective symptoms of the traumatic neuroses, inasmuch as the ingestion of about one hundred grains of grape sugar produces a noticeable glycosuria in from two to six hours. But as this symptom occurs under other conditions also and is not constant in the neuroses of traumatic origin, it is doubtful whether it is of much diagnostic importance (Strauss, Arndt, and others). An important point is the fact that the symptoms often extend over the *whole side of the body* on which the accident occurred. If it is, for example, an injury of the arm, and if this is found in a condition of contracture and paresis, the *cyanosis* and *atrophy* which take place may arouse a certain suspicion, as both phenomena, when not very marked, may be due to the inactivity, which may also be simulated.

If we, however, examine the leg which the patient can move, and of which he does not complain, we will often find the same phenomena, though to a less extent. It is to be especially noticed that after long standing only the leg of the diseased side assumes a *cyanotic color* and *feels cooler* than the sound one.

In examining the *sensibility*, all suggestive influence should be avoided. Instead of immediately asking the patient whether he feels or does not feel, observe first the condition of the *reflexes* and *movements of repulsion*, particularly upon painful stimulation. The examination may be very valuable in cases of unilateral sensory disorders where the other side can be used for comparison. If repeated examinations show that the reflexes are continually weaker or absent on one side, a disorder of sensibility upon this side is probably present. Next, the various forms of stimulation may be used, never forgetting to vary them, using strong and then weak stimulation, and comparing one place with another, particularly those places where sensation is normal, but slight with those areas which are endowed with a finer sensation.

If we have, for instance, a slight hypesthesia to touch, it will be particularly noticeable upon the slightest touch of a camel's-hair brush, while stronger ones will still be felt; or, if an hypesthesia of the leg is present, stimulation of the sole of the foot, of the balls of the toes, which have been even found decreased in healthy persons, is not felt at all, and at other places with less intensity, as comparison with the other side shows. The examination of the temperature sense after Goldscheider's method is also adapted to test the truthfulness of the patient, but consumes much time. But the following method, suggested by the same author, may be used with advantage:

> "In an apparent hemianesthesia or distinct limitation of a sensory in contrast to an anesthetic area, a long faradic brush is to be placed upon the borders of an analgesic area, so that it partly covers the sensory area, partly the part which is without feeling. A large place can be used as an indifferent electrode. Then without the patient perceiving it, the half of the brush should be lifted,—the one which covers the feeling part; the pain must then decrease or stop; upon lifting the half of the brush which covers the anesthetic part, the pain should become more intense, because the thickness of the current increases in the sensory part."

Gross contradictions in the answers of the patient render the result worthless; they may directly reveal his untrustworthiness, though it should be remembered that contradictions in the answers often occur in sensory examinations even in individuals who are suffering from organic diseases of the nervous system (tabes, etc.), so that one should be very careful in regarding them as being due to simulation. The fact that tests with a needle are not identical with those obtained from electricity must again be referred to. The results obtained from these methods need not be identical. Painful stimulations which are, unknown to the patient, suddenly applied to an analgesic place may evoke a lively movement of repulsion, because the suddenness of the stimulation, even though it is felt only as a touch, may excite terror.

It cannot always be decided with certainty whether an individual actually has pain or simulates it, the less so because this, as all other symptoms in the neuroses, is under the influence of the attention and self-observation of the patient, and decreases when his attention is distracted from it. The existence of painful pressure-points may be recognized in that pressure upon them entails not only pain but also a pronounced acceleration of the pulse (*Mannkopf's symptom*), or it is a painful position of the affected part of the body which causes an increased frequency of the pulse. Upon some occasions I was able, by exciting these pains, to evoke vasomotor phenomena,—*e.g.*, reddening of the corresponding side of the face. Or it is the painful movements and the tremor accompanying them which reveal the pains. In contrast, I have several times found that strong faradic stimulation with a brush, applied to the feeling parts of the body, strongly accelerates the pulse, and does not produce an increase worth mentioning when applied to the anesthetic parts. The negative result of these tests, the absence of Mannkopf's symptom, etc., should not without further data be regarded as a determination of simulation. The methods advocated by Fuchs and others for the detection of simulation of tremor are not, in my estimation, practicable. Many phenomena, which may be very troublesome, cannot be subjected to objective tests, as, for instance, the very common nervous tinnitus aurium.

Difficulty is offered by those cases in which the subjective troubles of the patient do not correspond to the objective examination. In such cases the physician should not be afraid to pronounce his "*non liquet.*" Another factor which may be very disturbing is the fact that among the laboring classes nervousness (and, according to Saenger, arteriosclerosis) has become more and more common, so that objective signs of this nature may have existed before the accident, and yet may wrongly be considered to be symptoms of a traumatic neurosis by the most observant physician. This error is difficult to obviate, but is not so very important, as in typical cases local relationships between the symptoms and the seat of the trauma are apparent.

It is dangerous to direct an examination towards ferreting out simulation without there being adequate reasons for doing so. Only when the result of the examination is negative or is contradictory and suspicious, is it advisable to direct your attention to simulation. In doubtful cases, however, it is recommendable to observe the patient in a hospital where the physician-in-charge is experienced in the determination of the neuroses and the psychoses. There sleep and the paroxysmal occurring phenomena (conditions of fever, spasms, and attacks of vertigo) may also be controlled. We must, however, agree with Freund, who says that it is a most difficult and tiresome task to differentiate satisfactorily a neurosis from simulation.

It is just as difficult to decide upon the *degree of disability* which is present. Exaggeration is often found in these cases, which may be pathologic (the patient actually overvalues his trouble) or is artificially evoked, so that one from the start mistrusts the patient.

If it is a purely local disturbance, only a decreased ability for work may be spoken of. But even where only a paralysis of an arm or leg is present, the general condition must be taken under consideration, as the signs of general nervousness may be present in addition to the local symptoms. If a condition is present which corresponds more or less completely to neurasthenia, a decision as to the ability for work would depend upon its severity. In cases of slight nervousness, work is generally a curative factor; a person who is suffering from severe neurasthenia is generally incapable of working, or can undertake only a modicum of work,—this is more true of the laboring man than it is of the teacher, merchant, etc. The same is true for severe forms of hysteria.

Though a patient can use any of his limbs, he is not necessarily capable of working. We must ask, How does the work influence his condition? Does he tire easily? Are movements painful? To answer these questions it may be necessary to ask the patient to work, observing the frequency of the pulse, respiration, etc.

I remember one case in which all the physicians had pronounced the patient fully capable of working, because he could move all his limbs freely, and when at rest was free from all trouble. But he insisted that he was unable to work, because he instantly became lame and dizzy. As a result of medical opinion, he was compelled to work. The local magistrate found him exhausted while working, and became convinced that he was incapable of working. He was therefore sent to me with the request to decide the question. I found at first no objective symptoms, but in lifting a burden—*e.g.*, an object weighing about fifteen pounds—the pulse rose from eighty to one hundred and twenty, and in carrying this burden the pulse and respiration increased gradually in so pronounced a manner that there remained no doubt of his disability.

It is better in doubtful cases to put the degree of limitation of the ability for work too high than too low; but, on the other hand, be careful in expressing an opinion of a complete incapacity. One should, especially in these conditions, not speak of a chronic disability.

It is apparent that one should be careful to discover whether the trouble existed before the injury and is deceitfully referred to this later accident. Alcoholism particularly may evoke phenomena which, in many respects, are identical with those of the traumatic neuroses. The detection of alcoholism, however, by no means indicates that the symptoms are entirely due to this intoxication. It must always be remembered that this factor increases the disposition to the traumatic neuroses, so that a relatively slight trauma (particularly a *cranial injury*) may cause a severe nervous trouble. It is not in such cases the duty of the physician to declare the patient's demand for a pension unjustifiable on account of the presence of alcoholism; he should only state the fact, should emphasize that alcoholism is present, but that it was the trauma which first evoked the nervous trouble, etc. Generally, however, in these cases also indications pointing to the traumatic origin of the disease may be found in the local symptoms caused by the injury (cyanosis, atrophy, local anesthesia).

A traumatic neurosis must often be differentiated from hematomyelia, vertebral caries, cerebral abscess, and other diseases under difficult circumstances. A careful examination and observation, however, generally render an error impossible.

Prognosis.—In mild cases complete recovery may occur, though those neuroses with marked local symptoms are often most difficult to cure. The position in life of the patient, litigation resulting from the injury, and too early resumption of work are all unfavorable factors.

The more the *psyche* is affected the less favorable is, in general, the prognosis.

If the cardiac and vascular symptoms are very marked and dilatation and arteriosclerosis are present, complete recovery is rarely possible. The prognosis is clouded further by the fact that this neurosis is often transformed into a psychosis. This circumstance has been observed by me in several cases,—for instance, in a case of mine which has been described by Goebel and which developed under my observation. The numerous cases of suicide which we find in recent literature upon the traumatic neuroses are probably referable to severe mental disturbance.

In those cases also in which local symptoms only were originally present I have often witnessed a marked change for the worse, even to such a degree that one could speak of a complete "nervous wreck."

Treatment.—The development of hypochondriac ideas should be prevented as soon and as much as possible, and the injured party should be assured that non-observation of his nervous troubles is the best measure for combating them. If one is convinced that an organic disease is not present, that the symptoms have developed from abnormal self-observation, it is advisable immediately to put the patient to work. Above all, it is necessary to give him time for recovery, and not to force him to a complete resumption of work at too early a period. This does not imply that he should rest until a complete return to health is secured, but his recovery can rather be hastened by prescribing physical exercise (gymnastics) carried on with care under medical attention. The proposal of Herzog, Strümpell, and others to test the ability for a partial capacity for work should be remembered.

The use of forcible measures is never advisable. For example, the attempt to reduce a contracture by force always leads to a worse condition. The same is true of the use of strong faradic-brush currents for the repression of tremor, spasms, etc.

In severe cases of general nervousness with depression of spirits, irritability, etc., a change of scene, particularly a stay in the country, may be efficacious. A cold-water cure also produces a good result. The baths of Cudowa, Nauheim, and Oeynhausen are likewise recommended. I saw improvement follow the use of the galvanic current in many cases and recovery in several. Galvanization of the brain and, with an existing stiffness of the back, the application of the current to the spine is recommendable. This treatment must extend over a long period of time, though I do not regard it as advisable to use it for longer than several months.

The faradic brush is often efficacious for the anesthesia. The paralytic conditions also, when not combined with contracture, may indicate the use of induction or labile galvanic currents. A mild massage exerts in such cases, as in those accompanied by muscular and articular stiffness, a favorable influence. The results achieved by the medico-mechanical institutions are not of the best.

If reflex epilepsy is present, excision of the cicatrix, from which the irritation arises, is demanded, but does not always lead to recovery.

Drugs are of doubtful utility; the ones used in the treatment of the common neuroses may be tried. *The psychic treatment* is the most important part of the therapy. The more the physician understands these patients and their troubles the more successful will be his treatment. The disease often recovers as soon as all litigation is over, in cases where damages have been asked for, but this is by no means the rule.

We should probably include among the traumatic neuroses those very bothersome sensations "in the missing part of a limb," which occasionally follow *amputations.*

The patient experiences pain and many sensations which he refers to the missing extremity. The condition is always a stubborn one. One of my patients, an Australian, had consulted the surgical authorities of all countries, and had many operations performed in order to secure freedom from his trouble; he had the constant sensation of his missing hand becoming clenched, spasm-like. All efforts for relief were in vain, however.

As in these cases there is no doubt that the trouble lies in mental acts which are not due to any organic cause, they should be included under the traumatic reflex neuroses.

HEMICRANIA (MIGRAINE).

This very common disorder occurs particularly in neuropathic individuals. Very often (according to Moebius, in ninety per cent. of the cases) direct heredity can be shown. The disease has been known to have descended through four generations, to have occurred in eight members of the same household, etc. It generally is first observed at puberty, often in early childhood, but it may develop at any time up to the prime of life, rarely later. Women are more often affected than men.

Heredity is the most important etiologic factor. Other moments are only exciting causes. Mental strain, continued emotional excitement, working in superheated rooms may, in predisposed persons, produce this disease. Masturbation has also been blamed. There can be no doubt that it may be evoked reflexly. Some observations indicate that diseases of the nasal mucous membrane (hypertrophy of the mucous membrane, enlargement of the corpus cavernosum) may particularly act in this way. Its origin from affections of the sexual apparatus and from entozoa, etc., is less firmly established. The relationship between gout and migraine has not been satisfactorily explained, but has been emphasized by Gowers and Charcot.

Individual attacks are particularly excited by emotional outbursts, alcoholic excess, and mental strain; coitus may also evoke an attack (Determann). Variations in atmospheric pressure have been blamed by Marcus.

The most important and often the only symptom of this disease is a periodically occurring severe headache which is generally combined with gastric disorders: loss of appetite, nausea, a feeling of strangling, and vomiting. The attacks last from about twelve to twenty-four hours, may be shorter (two to three hours), or may last from two to three days.

The headache does not generally commence suddenly in full intensity, but prodromes precede it. These consist of a feeling of apathy, sleepiness, a tendency towards yawning, a feeling of fulness of the head, vertigo, depression, etc.

One of my patients had a boulimia in the evening before an attack, another became very excitable. The headache is dull at first, and gradually increases in intensity until often it becomes so severe as to be unbearable. It does not always confine itself to one side of the head,—as one might think from the word hemicrania,—though the left side is the most often affected. It may, however, involve the entire frônt part of the head or the frontal or temporal region of both sides, or is felt, now here, now there, most intensely. It may also invade the occipital region. It begins sometimes upon one side and extends to the other; patients have upon several occasions assured me that the seat of the pain regularly alternated in recurrent attacks.

An hyperesthesia of the skin of the head or a sensitiveness to touch of the emerging branches of the fifth nerve is sometimes present during an attack.

During the pain the patient feels limp and miserable, and is abnormally sensitive to stimulation of the special senses. He cannot bear a bright light or noise or a strong smell. In order to avoid these he darkens his room and excludes himself as much as possible from the outside world.

Movements of the head or eyes increase the pain. There is complete loss of appetite, and if *vomiting* occurs it may come on during or at the end of an attack; diarrhea and polyuria may also occur towards the end of an attack. *Ptyalism* rarely occurs during a paroxysm. Dacryorrhea and hyperidrosis (Liveing) are also rare symptoms.

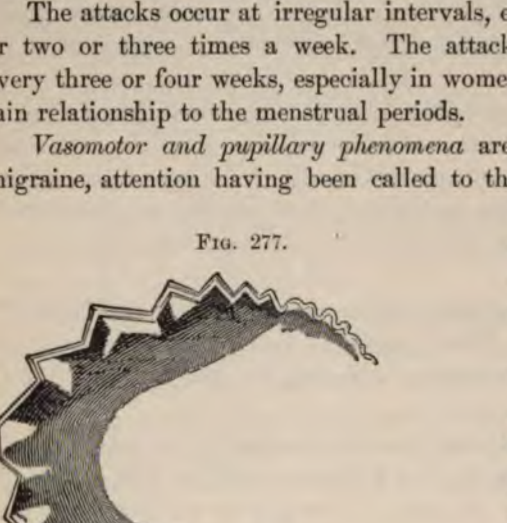
Sleep generally brings on the crisis, the patient awakening with a clear head and feeling well. If the attacks are severe, coughing, straining, sneezing, etc., may cause a momentary headache for some days after the attack has ceased. Slight attacks are sometimes aborted by work, the ingestion of food (Moebius), etc.

The attacks occur at irregular intervals, every few weeks or months, or two or three times a week. The attacks may come on, however, every three or four weeks, especially in women, in whom they bear a certain relationship to the menstrual periods.

Vasomotor and pupillary phenomena are inconstant symptoms of migraine, attention having been called to these symptoms by du Bois-Reymond, and are due to excitation of or paralytic conditions of the sympathetic.

In one case the face blanches, the skin becomes cool, the bitemporal artery contracts and hardens, the pupils dilate, the salivary secretion increases; in another case the face and conjunctiva flush, the artery dilates and the pupils contract, and unilateral hyperidriosis may occasionally be found. These phenomena have given rise to the conception of two forms of hemicrania: *hemicrania sympathicotonica, or angiospastica*, and *hemicrania sympathicoparalytica.* Typical cases, however, rarely occur. Symptoms of irritation and of paralysis may exist conjointly or pass into each other, and in many cases the pupils and facial color are not altered. The few observations of this kind are not at any rate sufficient to regard hemicrania as being due to disease of the sympathetic.

FIG. 277.



Scotoma scintillans. (After Charcot.)

There is a class of cases, however, in which the migraine attacks are accompanied by *disorders of function of the cranial nerves*, by *anomalies of sensibility and of motility*, and also by *speech disturbances*.

The most common are the cases of *ophthalmic migraine*. The attack is ushered in by a flickering before the eyes, generally combined with some disturbance of vision. A bright spot appears at some place in the visual field, and extends or is transformed into a bright irregular (zigzag) figure which gradually fills the entire visual field with a glaring, flickering light, sometimes of many colors (Fig. 277). The disorder of sight which occurs at the same time is of the character of a *scotoma* (scotoma scintillans) or a partial or complete hemianopsia, more rarely an amaurosis. All these phenomena, which may vary in many ways, last but a short time (a few minutes to half an hour); then comes the headache. In a few cases the individual loses all power of speech during an attack.

The *aphasia* is rarely, however, complete, and, as I observed in one case, may be combined with agraphia. Word deafness has only been observed in a few cases. This disturbance lasts from a few minutes to half an hour, and the headache which succeeds it is always upon the left half of the head. Hemianopsia and aphasia may also occur simultaneously.

I treated a man who suffered, as did three of his brothers and sisters, from childhood, from ophthalmic migraine with aphasia. An hereditary taint was not present, but the father worked with potassium cyanide.

Paresthesias in one arm or upon one-half or upon both sides of the body (for example, upon the lips, tongue, etc.) may be accessory symptoms of a migraine attack. An anesthesia corresponding to it cannot often be detected. A feeling of *weakness* in one arm or in one half of the body, which corresponds to a real paresis, may also constitute one of the focal symptoms of migraine. If it affects the limbs of the right side, the head-pain is localized upon the left half of the head.

A case observed by me differed from all others which have been described. In it each attack of migraine was accompanied by typical cerebellar symptoms. During an attack the station and gait were very uncertain. The patient reeled like a drunken person, was affected with pronounced vertigo, and had a sensation of his body or parts of it being double. The incoördination commenced with the onset of each attack, to disappear at its termination. This case could be described as one of *cerebellar hemicrania*.

Hemicrania may also be combined with psychic disorders (Griesinger, Krafft-Ebbing, Mingazzini). It is generally a transitory insanity, a condition of excitation, confusion, or stupor often combined with hallucinations, which soon passes away. Mingazzini styles this psychosis transitory hemicranial dysphrenia (dysphrenia hemikranika transitoria).

The disturbances described above may be combined in many ways. They sometimes repeat themselves in a stereotype manner in every attack, but there is often much irregularity in regard to these complications in that they may appear in one paroxysm in full intensity to be absent or incomplete in another. It is not uncommon for an individual who has suffered from simple migraine for a long time to become afflicted with these complicated attacks later on.

Those suffering from migraine do not present any objective symptoms of disease except during the attacks. The disease, however, so frequently accompanies *neurasthenia* and *hysteria* that symptoms of these neuroses are found, often during the intervals also. A condition of general nervousness develops almost always if the disease has existed long and the attacks occur often and are severe. The patient has then the look of a sufferer, and becomes gray and aged early in life.

Hemicrania also associates itself with other diseases of the nervous system,—for example, with *writer's cramp, convulsive tic*, etc. Its relationship to epilepsy is of special interest; this has been particularly treated by Liveing, Gowers, and Moebius. Both diseases may occur conjointly, or the phenomena may occur so quickly after each other that their separation is difficult. A person who has been suffering for a long time from hemicrania may also become epileptic.

Aside from these combinations, the relationship between these two neuroses cannot be mistaken. In both, the nervous symptoms come on in paroxysms, with auræ often preceding the attack and sleep tending to terminate it. *Cardialgia* or *severe vertigo* may occur vicariously for the migraine attack (hemicranial equivalents, Moebius), just as we have equivalents of an epileptic attack. Moebius regards gastroxynsis as a variety of migraine. I have observed cases in which severe pains occurred on circumscribed parts of the body or extremities in place of true hemicrania, and which disappeared spontaneously after some hours or a day to reappear in a few weeks in a similar manner. Lamaq has confirmed this observation. In another case, attacks of *hemiparesis* vary with the migraine paroxysms.

It is noteworthy that a *paralysis of the ocular muscles* may also occur in migraine, the so-called *periodic oculomotor paralysis* (page 284) being closely related to this disease. In individuals troubled with migraine I have repeatedly observed a *persistent paresis* of one or more eye-muscles (abducens paresis, ptosis, internal ophthalmoplegia), without being able definitely to determine whether it was an uncommon symptom of this disease or the prodrome of an organic brain disease. In those persons whose migraine was always on one side I occasionally found the palpebral fissure and pupils of the affected side chronically contracted.

It is doubtful whether *psychic disturbances* may occur as equivalents of hemicrania, although the psychic disturbances which develop in patients suffering from migraine and apparently replace the paroxysms of pain seem to denote this. These include attacks of mania, acute mental confusion, and, in one of my cases, melancholia.

In hysteric persons who were sufferers from migraine I have observed in several cases during the attack a *hemianesthesia* with disorders of the special senses upon the side corresponding to the seat of the head-pain.

There is a form of migraine which is characterized by the *constancy of the head-pain.* It might well be called a permanent hemicrania. Moebius speaks of a status hemikranicus. Its chief point of differentiation is that it occurs in persons who have suffered from typical attacks for a long time, or whose parents were troubled with true migraine. The conversion of migraine attacks into this permanent form has been observed by me, particularly in *neurasthenic* or *hysteric* persons.

In contrast to these cases are the partial attacks which represent an abortive form of true migraine, a simple flickering before the eyes of short duration, or a temporary nausea, with slight intracranial pressure, etc., representing the entire attack.

Pathology.—It should not be assumed that this disease has an anatomic disease as its basis. Many facts speak for a *vasomotor* origin for the migraine attacks; not so much the vasomotor disturbances occasionally accompanying them, which may be the effect of the pain, as the *cerebral symptoms* described above, whose transitory nature indicates a nutritive disorder which can best be explained by referring it to a *vascular spasm.* These cerebral symptoms precede the attacks of pain, and therefore cannot be regarded as results of it. The cerebral membranes are probably the seat of the pains.

The theory of a vascular spasm also sheds light upon the fact that in some cases the transient phenomena of a complicated migraine attack (hemianopsia, aphasia, etc.) may become chronic symptoms (Charcot). In one of these cases I found a thrombosis of the *internal carotid* (just before the giving off of the middle cerebral artery) which had produced the paralytic symptoms.

Diagnosis.—In typical cases, the disease cannot be mistaken. A headache combined with vomiting caused by a cerebral tumor or by uremia may, it is true, be confused with migraine, but a careful examination should prevent this error. Though the headache in cerebral tumor may also appear paroxysmally in the initial stages, other symptoms will always be present upon which a diagnosis can be based. It must be remembered that retardation of the pulse occurs also at the height of an attack of migraine; it sank in one of my cases to forty-eight beats a minute. Hemicrania in children may be difficult to diagnose. I have seen such cases diagnosed as cerebral tumor or as meningitis. The periodicity of the headaches, the regular length of single attacks, and the absence of objective symptoms in the intervals should, however, prevent errors of this nature. The determination of the existence of hemicrania in one of the ascendants should help in establishing a diagnosis.

Course and Prognosis.—The disease is chronic. It may disappear at the climacteric or in old age. A cure in early life, either spontaneously or from treatment, is rare. Pregnancy, the onset of menstruation, or a change of climate may, however, effect a cure. In one case, the hemicrania disappeared after a cranial injury. In a patient who had suffered from early youth with hemicrania, the headache disappeared, after an attack of typhoid fever, for twelve years. A gentleman who consulted me on account of neurasthenia had migraine attacks from childhood until his thirtieth year, from which time they failed to appear. His physician had assured him that the trouble would cease at that time. In some cases the vomiting accompanying the attacks ceases to come on after some years; in one of my patients the hemianopsia which had previously accompanied the attacks disappeared.

We sometimes observe a transformation of the hemicrania into epilepsy. Hemicrania may also be a *prodrome of tabes dorsalis and of paralytic dementia.* The migraine disappeared when the tabes developed or the attacks were transformed into gastric crises. As to the connection of hemicrania with paralytic dementia, the form combined with cerebral symptoms of degeneration deserves in this respect some attention, inasmuch as they constitute in some cases a prodrome of paralytic dementia. Hemicrania is rarely curable. Life is, however, hardly endangered, if we exclude those very rare cases in which the vascular spasm leads to thrombosis. The prognosis is somewhat clouded by the tendency of migraine to combine with hysteria, neurasthenia, epilepsy, etc., and—in a few instances—to pass into tabes dorsalis and paralytic dementia. In this respect the cases of ophthalmic migraine are to be feared more than the other forms, when it comes on only in later years. In most cases, however, this form is also harmless.

Treatment.—The factors which may induce an attack of migraine must be relieved in every case, and the treatment should, as much as possible, be adapted to this purpose. Injunctions as to the manner of living, the diet, etc., for instance, may do more good than drugs. The patient himself generally learns to avoid those injurious things which evoke an attack.

In many cases the patient is suffering from obstinate *constipation*, and regulation of the movements of the bowels may do much good; excellent results, for instance, have been achieved by the use of Carlsbad water or salts corresponding to it, or by the use of cold-water clysters. Treatment of chronic nasal diseases has also partly relieved and even cured the attacks in some of my cases. *Tonsillotomy* of a hypertrophic tonsil cured one of my cases. If *anemia* is present, it should be appropriately treated with iron. Change of climate, and especially a long sojourn in the *mountains* or by the *sea*, may be of much benefit. These therapeutic procedures are intended to improve the general condition. In this sense *hydrotherapeutic measures, general massage, gymnastics, galvanic, faradic,* or *static currents*, general faradization, or galvanic currents to the head, etc., are general methods of treatment that should be used in every case, though the results are not brilliant.

Arsenic is the drug of most value. It has produced cures in some and betterment in many of my cases. In one case, in which the attacks were so severe that the patient threatened suicide, all remedies proved useless, until by the use of Levico (arsenical) water I procured a cure; he had lost so much flesh from the severe attacks that in the first four months of the time that he was free from paroxysms he gained ten pounds in weight. Arsenous acid is probably the best form of arsenic to use. The waters of Levico, Roncegno, and Guber may also be prescribed. It is often advisable to prescribe iron at the same time.

Charcot recommended the continued use of the *bromides*, in increasing doses, as does also Moebius, who uses sodium salicylate when the bromides cannot be borne.

Gowers praises *nitroglycerin* in the angiospastic form in doses of from one-two-hundred-and-fiftieth to one-hundredth of a grain three times a day, prescribed in tablet form or in an alcoholic solution (one per cent.). The dose of this is one drop; one can add some hydrochloric acid or tincture of nux vomica to it.

Most of the drugs used in migraine serve to *combat single symptoms*. It is true that a number of drugs are capable of shortening an attack, of lessening the intensity of the pain, or of completely annulling it. Every drug, however, has not the same influence upon all individuals, and there is none whose efficacy is not lost in time.

Sodium salicylate, two to three grams (thirty to forty-five grains) in water or in a cup of strong coffee, has a pain-stilling action. So have antipyrin,[1] from five to fifteen grains (0.5–1.0), phenacetin, ten to fifteen grains (0.75–1.0), caffeine citrate, two grains (0.15), or caffeine and sodium salicylate, three grains (0.2). In some cases antifebrin, from four to five grains (0.25–0.3), exalgin, four grains (0.25), analgen, from five to fifteen grains (0.3–1.0), methylene blue, one and a half grains (0.1), pasta guarana or paullinia sorbilis, from thirty to sixty grains (2.0–4.0), and amyl nitrite (a few drops upon a handkerchief) are also recommended in the spastic form; and ergotin (extract of ergot aq., 2.5; spirit. dilut. and glycerin, āā ʒi gr. 15 (5.0). S.—Use one-sixth to one hypodermic syringeful as a dose), lately cytisin, one-twentieth of a grain (.003–.004), and migränin, sixteen grains (1.1) to a dose, in the paralytic form. I cannot assert that migränin justifies its name.

---

[1] This drug is probably praised most by those suffering from hemicrania; I have seen a paradoxic action from its use: it increased the pain and the number of attacks.

I have found potassium bromide in combination with caffeine efficacious in some individuals.

A cold compress, firm compression of the head, menthol rubbed over the frontal and temporal region (in alcoholic solution, 3.0 to 20.0 (forty-five grains to five drachms) of alcohol, or in the form of a pencil) may relieve the pain. A hot foot-bath or a mustard poultice to the nuchal region may do good. Instillations of cocaine into the conjunctival sac relieved one case. Morphine must not be prescribed unless absolutely necessary.

The use of electricity during an attack does no good. To static sparks have been given the credit of having aborted attacks in several cases (Determann).

Many patients refuse all treatment, remain quietly in their darkened rooms, take nothing except a little tea, seltzer, lemonade, etc., and bear the pain with patience.

HEADACHE (CEPHALALGIA, CEPHALÆA).

Headache is a symptom of many diseases of the nervous system and of other organs, and only rarely has the importance of being an *independent disease*. The form occurring in organic cerebral diseases will be discussed here only in so far as is necessary for differential purposes. Hemicrania was discussed in the previous chapter.

The *headache* is often caused by *circulatory disturbances* within the cranium. Hyperemia, anemia, and particularly variations in intracranial blood-pressure may evoke it. *Active hyperemia* causes, as a rule, a severe, beating, often pulsating headache, which is frequently combined with vertigo, tinnitus, flickering before the eyes, flushing of the face and of the conjunctiva, etc. In some individuals this condition arises from *primary vasomotor* disturbances (*vasomotor cephalalgia*). From time to time these attacks of vascular congestion come on; the face and ears become red, the skin feels hot, and the pulse is full and rapid. It is evoked—and also the single attacks—by mental strain, emotional excitement, alcoholism, excessive smoking, onanism, and injuries to the head. *Venous hyperemia* of the brain is an important cause of habitual headache, especially the severe forms as seen in heart disease, pulmonary emphysema, etc. Forced continued coughing, even compression of the veins of the neck by a tight collar, may act in this way.

Anemia, whether due to chlorosis or to loss of blood, is accompanied by headache. This is generally dull and heavy, rarely severe, and is found in front or on the side of the head, or over the whole head. Headache due to hyperemia is increased by stooping, coughing, sneezing, etc.; the anemic form is generally relieved by assuming a recumbent position.

The headache so often accompanying *arteriosclerosis* is probably also for the most part a product of *circulatory* and *nutritive* disturbances, although it is possible that the rigid vessels irritate the meninges, thus causing headache.

In not a few cases the cephalalgia is of *toxic* origin, as in acute *alcoholic poisoning*, in *nicotine*, *caffeine*, *morphine*, *chloroform*, and *ether* intoxications, and from the action of metallic poisons.

Gastric disorders (indigestion, gastric catarrh, and *obstipation*) also produce headache, due probably to autointoxication. Reflex causes may also cause headache, as in the headaches due to *menstrual disorders*, *worms*, etc.

Headaches due to fever, uremic, acetonemic, and diabetic headaches, etc., are probably also due to *autointoxication*.

A severe headache may occur in all the infectious diseases (typhoid, influenza, malaria).

Rheumatism of the frontal and occipital muscles produces a boring pain, which is increased by pressure and movement of the skin of the head.

Diseases of the cavities of the skull and the mucous membranes clothing them (nasal, frontal, pharyngeal, tympanic cavities) may also cause headache. Errors of refraction, especially hypermetropia and errors of accommodation, may also be causes. In some cases every persistent effort at fixing the eyes, or every time they are strained, results in cephalalgia. Sexual excesses, especially masturbation, may produce headache.

In a majority of cases the headache occurs from *neurasthenia*, *hysteria*, and *hemicrania*. Even when it is apparently the only symptom, or all that the patient complains of, is this often the case. The symptoms of a neurosis may remain latent for a long time, only one symptom appearing. Careful observation will, however, generally show the neurasthenic or hysteric nature of the trouble. In some cases, however, there is no sign of a general neurosis, and the *headache represents the disease. Heredity* is, however, often in play. Hemicrania may produce a constant headache, and may also cause some type of headache in the offspring. Charcot speaks of a headache of puberty (cephalæa adolescentium) as an independent disease.

Mental overexertion, working in *overheated* rooms, trauma, staying up at nights, etc., are also factors. Cranial injuries which have not directly involved the brain and its membranes may also result in headache (particularly the vasomotor form).

The characteristics of neurasthenic, hysteric, and hemicranial headaches have already been described in their appropriate places. *Habitual headache* may be periodic in its appearance, but not at regular intervals; the individual attacks are also of variable duration, in many cases being described as a permanent, only partly remittent pain. If severe and chronic, it influences the general condition, produces depression, makes the patient appear prematurely old, and excites hypochondriasis.

The prognosis depends upon the cause. Habitual, primary headache is not rarely a chronic trouble that lasts for years, even for life.

The recognition of the cause is the prime essential for a successful **treatment.** One should not be satisfied with merely making a diagnosis of cephalalgia. A careful examination of the entire body, a minute dissection of all possible factors, is necessary to achieve success in the treatment. Symptoms of cerebral tumor, of cerebral lues, of meningitis, etc., must be sought after. The cranial cavities must be examined, the special senses tested. Errors of refraction must be corrected. The condition of the heart and vessels and of the urine may furnish important points. *Hydrotherapeutic measures* are often beneficial in cases of congestive cephalalgia, particularly cold rubs, lukewarm half-baths, warm foot-baths, hot foot-douches, wet packs around the feet, etc. These procedures may also relieve and cure *neurasthenic headaches.* Electricity, a stay at the sea-side, in the high mountains, or merely a change of scene, or perhaps a sea-trip or a journey to the South may also be prescribed for this and similar forms of headache. *Psychotherapy* is very beneficial in such cases.

More simple measures, as a cold compress, menthol pencilling, massage of the nuchal region, inhalation of sal ammoniac, washing with chloroform, etc., are known to the patient, and in mild cases may do good. Rheumatism of the muscles of the head may be successfully treated with diaphoresis, massage, and local faradization.

The preceding chapter should be consulted for the drugs that may be used. Hardly one of those mentioned there has not been used in other forms of headache also and has occasionally exercised a curative influence. I have had no success with methylene blue, a drug that has been praised by others, but have seen *pyramidon* do much good. The narcotics should be prescribed cautiously, and only when absolutely necessary.

There are some particularly stubborn cases in which none of the measures advised above do any good. In such cases a *seton* or a *cautery* may be used with benefit. I observed in some cases headache which had existed for years, often preventing the victim from working, completely disappear after the application of a seton.

VERTIGO.

This is a symptom of uncertain value, as it occurs in the most different forms of disease and may be an important symptom or merely a general phenomenon of doubtful value. But the fact that it may be the *only* or the chief symptom we consider a sufficient reason for discussing it separately.

A common *cause* is the false projection of the visual field in paralysis of the ocular muscles. It accompanies, in these cases, diplopia, but may be absent or soon disappear. The view that vertigo always results from an altered function of the ocular muscles is certainly not correct. This opinion is due to the fact that the oculomotor nucleus, which, according to the investigations of Shimamura and d'Astros, is not well supplied with blood, is most sensitive to every disturbance of the intracranial circulation.

Unaccustomed positions, and especially rapid revolving of the body, or swinging, or looking down from a high elevation, produce a feeling of vertigo in most persons. Nausea and vomiting generally accompany attacks of severe dizziness. It is a common symptom of organic brain diseases which produce an *increase in intracranial pressure,* particularly of tumors.

This is especially true of *cerebellar tumors ;* all lesions of the cerebellum, however, cause vertigo.

Diseases of other parts of the brain which take part in co-ordination may, however, also cause vertigo ; as of the medulla oblongata, cerebellar peduncle, and vestibular nerve. The occurrence of hemorrhage or softening in any part of the brain may reveal itself by an attack of vertigo, which, as a rule, is not repeated. Every sudden *alteration of the circulatory conditions* in the brain may act in this way, as may also diseases of the blood-vessels, accompanied by a chronic retardation of the intracerebral circulation, as in *atheroma*. In these cases we find single vertiginous attacks and also a persistent feeling of sinking, of dizziness, and of stupor.

Acute anemia as well as congestive hyperemia of the brain may produce attacks of dizziness. Women at the *climacteric* frequently suffer from this condition.

Vertiginous attacks are an almost constant symptom of *multiple sclerosis*. They are generally sudden seizures of more or less incoördination, even so severe that the patient falls to the ground; we find more rarely a persistent condition of dizziness. It may be an equivalent of *epileptic attacks*, and may also occur vicariously for migraine.

Alcohol, *nicotine*, and *caffeine* are the chief poisons of practical importance that produce vertigo. Uremic vertigo is due to autointoxication. Indigestion and overloading of the stomach may produce vertigo. Obstipation is a frequent cause, also cephalic injuries. Intestinal parasites may sometimes evoke them, and removal of a tapeworm may relieve them.

The forms of vertigo occurring with *diseases of the ear* are of much practical interest. Every disease of this organ, even a collection of wax in the external auditory canal (Toynbee), may produce it. There is a particular group of cases in which the vertigo is in almost constant combination with aural symptoms, and in such a typical manner that Ménière (in 1861) classed them as a separate disease.

In Ménière's disease the vertigo occurs suddenly and paroxysmally, the patient falling to the ground as if shot, stricken dumb, or even momentarily unconscious. During the vertigo, he thinks he is revolving in a circle, or feels as if the surrounding objects were moving, or as if the floor were sinking, etc.

Adler and Guye observed that in unilateral diseases of the labyrinth the vertigo was associated with a feeling of moving towards the diseased side, and that in turning his head or body towards this side a revolving vertigo occurs in which the patient imagines the surrounding objects to turn in the same direction, phenomena which do not occur if the movement is made towards the sound side. Adler considers this to be due to an over-stimulation of the diseased organs of co-ordination.

Nausea and *vomiting*, which may last for hours, and headache generally accompany the attack (Jackson, Lucae, Schwabach). It is more rare to see a facial paresis come on during an attack (Charcot, Frankl-Hochwart). I have noticed the same. The patient appears pale, collapsed, and the skin is covered with a cold sweat.

The auditory accessory symptoms are characteristic. There is *difficulty of hearing*, as a rule, with decrease of osseous conduction on one side; also a *stubborn tinnitus aurium* is present.

Before the vertigo comes on, the roaring becomes severer, higher, and shriller. This is, however, not always the case. *Nystagmus* and diplopia also occur occasionally. The attacks may repeat themselves daily, or at intervals of weeks or months; at a later period of the disease a chronic vertigo manifests itself, which only occasionally undergoes exacerbation (particularly in coughing, straining, etc.). The disturbance may be so great that the patient is afraid to leave his bed. In the cases first described by Ménière, the disease came on suddenly in persons whose ears were until then perfectly healthy. The designation Ménière's disease is, however, now applied to cases in which the phenomena occur in persons who were previously troubled with ear-disease, and where they come on slowly. V. Frankl believes that in such cases we should speak of Ménière's symptoms and not of Ménière's disease. Even though this symptom-complex may be found in middle-ear diseases (Gellé), and in a few cases even in diseases of the external ear, the clinical phenomena, as well as the post-mortem results (Ménière, Politzer, Gruber, Moos, and others), point to a labyrinthine seat for the disease. In the typical cases it appears to be due to a hemorrhage in the labyrinth. It may occur in otherwise healthy persons or be due to syphilis, gout, leukemia, and other general diseases. Injuries (*e.g.*, fracture of the petrous bone) and inflammations may also cause it, whether primary or secondary. The labyrinthine inflammation may arise from the middle ear, may be of syphilitic or of an epidemic-meningitic origin. Gruber emphasizes the fact that it is particularly the exudation processes in the adnexa of the labyrinth (recessus Cotugni) which may lead to an increase of the endolymph or in cases where there is a displacement of the outlet of the *saccus* to an impaction in the labyrinth, and thus produce the symptoms of Ménière. A lesion of the cochlea produces deafness; a lesion of the semicircular canals, probably, vertigo and incoördination. The intimate relationship between the vestibular nerve and the nuclei of the ocular nerves probably explains the ocular symptoms (P. Bonnier). I have rather often observed Ménière's disease in persons suffering from atheromatous arteries, and in one which came to a post-mortem, I found that the arteries passing to the ear were diseased. Cranial injuries may likewise evoke this disorder.

Disorders allied and similar to Ménière's symptom-complex occur in hysteria and neurasthenia, and also as an aura of epileptic attacks (pseudo-Ménière, according to Frankl). These are probably due to vasomotor disturbances, as is also the toxic form of *aural vertigo*. Ménière's symptom-complex has also been observed in tabes. It has not yet been determined whether a primary acoustic disease may evoke this disease, though I think that it is probable. Some persons become dizzy from hearing high-pitched tones. Vertigo is less often found in connection with *nasal diseases*, but it does occur in such conditions, and is relieved when the original trouble is cured. There occurs also a *periodic swelling* of the nasal mucous membrane due to vasomotor disturbances, which may provoke vertigo.

A *laryngeal vertigo* has also been described. This is a rare phenomenon, and hardly justifies the term vertigo. A person with some lingual trouble suddenly becomes partially unconscious, with apoplectic or epileptoid symptoms, followed by a burning in the larynx and sometimes by coughing.

Vertigo is often a symptom of nervousness, particularly *hypochondriac neurasthenia*, and there is hardly another symptom so readily produced by introspection and self-observation. It is my belief that every one may produce in himself a feeling of dizziness by thinking of incoördination and by depicting to himself the feeling. This is to a greater extent true for neurasthenic individuals, in whom fear of vertigo and thinking of it are sufficient to cause an attack.

An endemic type of severe vertigo, appearing in the Geneva canton, Switzerland, with a kind of paralytic weakness of the extremities and of the muscles of the nucha, together with ptosis (occasionally disorders of vision, dysphagia, etc.), has been called *paralytic vertigo*, and was supposed to be due to miasmatic influences (Gerlier). The affection occurs in paroxysms, the individual being healthy in the intervals. The nature of this disease has not yet been discovered. I spent some time in Geneva studying cases of it (assisted by Dr. Ladame), but to no purpose. Miura, in Japan, has lately described a disease as *kubisagara*, which is probably identical with Gerlier's vertigo.

The prognosis depends upon the character of the original trouble. *Vertigo* due to a diseased stomach is almost always curable. The vertigo of arteriosclerosis may disappear later in the disease. In most cerebral diseases it is, however, incurable. Ménière's disease is occasionally cured or improved, but is generally very resistant to treatment. Recovery and deafness sometimes come on simultaneously. The prognosis of nasal vertigo is favorable.

Treatment.—The cause should be remedied if possible. If due to cerebral hyperemia, it may be cured by *purging*, *hot foot-baths*, and *bloodletting*.

Gastric disorders must always be treated and relieved in cases of vertigo. *Tænia* must also be removed from the system. In cases of arteriosclerosis, potassium or sodium iodide should be given. The combination of the iodides with ergotin has also been recommended. It may be combined with the bromides if a symptomatic action is desired.

In Ménière's disease, the aural disease must be treated. The vertigo may often be relieved by the use of the bromides or of belladonna. Better results, according to Charcot, may be expected from the administration of *quinine*. It must be given in daily doses of from twelve to fifteen grains for from two to three weeks, then discontinued for some time, to be renewed again later. At first it may make matters worse (the quinine, according to Charcot, is struggling with the disease); afterwards a steady betterment is noticed. I have seen it do good in such cases, though aurists are opposed to its administration, fearing that it may increase the deafness. Charcot also recommended puncture of the tympanum for its relief, as the vertigo disappears when the deafness comes on. This is, however, no pleasing inducement to the patient.

I have seen bloodletting do good in several cases, particularly in patients in whom the attack was accompanied by symptoms of congestion. *Sodium salicylate* has been highly recommended. Hirt praises *pilocarpine injections* (five to eight drops of a two per cent. solution every second day, continued for weeks). Gruber prescribes the tincture of arnica with the tincture of nux vomica.

Treatment of the hypertrophy of the nasal mucous membrane may be beneficial.

If the symptoms are of neurasthenic origin, the treatment should be similar to that of neurasthenia. To combat the symptoms one may try the bromides, phenacetin, hydrobromate of quinine, nitroglycerin, galvanization of the brain, faradization of the back of the neck or the soles of the feet. Static electricity may be used in a similar manner upon distant parts of the body. In some cases of mine of very persistent vertigo, hot foot-douches were found efficacious. The use of a seton may relieve after everything else has failed. The disease may, however, resist all treatment.

GLOSSODYNIA (NEUROSIS OF THE TONGUE).

This symptom—we cannot call it an independent disease—consists in paresthesias, particularly in a burning and prickling sensation, on the tongue, extending perhaps to the adjacent mucous membranes. It may occur paroxysmally or continuously, and may even disturb sleep. It has been observed particularly in women of advanced age, but is not very common. The teeth were almost always absent. A neuropathic predisposition was generally present. A gouty diathesis may also tend to cause it. *Cancrophobia*—fear of cancer of the tongue—was often present. It could not be decided, however, which was cause and which effect, whether the hypochondriasis evoked the paresthesias, or *vice versa*.

We have different types of epileptic attacks: (1) major epilepsy, or attacks of *grand mal;* (2) milder types, or attacks of *petit mal;* (3) the *equivalents* of epileptic attacks.

Complete attacks, or *grand mal,* may come on suddenly. In many cases premonitory symptoms are present, as vertigo, depression, trembling, irritability, transient muscular twitchings, coming on in an extremity or diffusely over the entire body (Reynolds, Charcot), and, more rarely, urticaria, photophobia, tinnitus, etc. More often, however, they are phenomena which occur some seconds or minutes before the attacks, or at the beginning of them. These are called *auræ,* and are varied in their nature, though each patient has the same sort of aura at each renewed attack. Some attacks also come on suddenly with unconsciousness, without any auræ preceding them.

The symptoms of the auræ may be of a *motor, sensory, secretory, vasomotor, visceral,* or *psychic* nature.

The motor auræ consist of twitchings, which involve, as a rule, one extremity, and perhaps only one muscular group of it, and may then extend farther in the same manner as was described under cortical epilepsy. Generally, however, unconsciousness and general spasms follow so soon after it that the aura consists only of a few twitchings confined to a few muscles or to one extremity. Symptoms of motor irritation occurring on both sides of the body may also precede the attack. Complicated movements, as running forward and backward in a circle, or scratching, etc.,—*i.e.,* automatic and complicated co-ordinated movements,—occur more rarely.

Sensory auræ are more common. They include paresthesias, formication, numbness, etc. These paresthesias may also extend upward and downward before consciousness is lost. A peculiar sensation passing upward from the lower abdomen or gastric region may be experienced. Globus may also be the aura of an epileptic attack. Sometimes it is a curious, strange feeling which the patient cannot describe. The aura may also consist of *pains,* generally a pain in the epigastric region or a painful sensation passing upward from the abdomen, and but rarely a dull headache.

A peculiar *taste* or *smell* or some *aural* or *facial* sensation often constitutes the aura. The auditory auræ consist in the hearing of a noise (whistling, roaring) or a clang, more rarely of a word or a melody, or in sudden deafness. The *visual* auræ consist in the seeing of colors or spots, or of a complete picture. A sudden loss of light may also constitute the aura. In other cases it appears as if objects decrease or increase in size, approach or recede, or as if they revolved. In these cases a feeling of vertigo is also generally present. The vasomotor auræ are characterized by a sudden pallor, which may be limited to one side of the body, or flush, or the appearance of circumscribed red spots.

A *secretory* aura is rare, and consists generally in a *profuse perspiration.*

Terror, shuddering, depression, gayety, rage, or recollections of some previous event in his life, etc., constitute the *psychic auræ.* Or there may be a sort of memory hallucination, in which the patient thinks he has lived the present once before. The psychic auræ may reach the intensity of a *pre-epileptic insanity*, resembling delirium.

The Attack.—Following upon the aura, or more often without any premonition, the patient suddenly falls *unconscious.* At the same time a *tonic, tetanic rigidity of the muscles* occurs, involving the respiratory muscles also, so that breathing stops. The sudden, forcible expulsion of the air with the simultaneous closing of the glottis produces a short shrill or muffled cry, which in many cases is the first signal of the attack. At times it is an inspiratory gurgling murmur.

The face at first pales, soon becomes red, then bluish-red, appears bloated, disfigured; the eyes are closed or open and fixed, and the pupils are dilated and do not react. The head is turned to one side or backward (sometimes the whole body turns); the eyeball is turned towards the same side or rolled upward, the arms are extended or flexed, while the thumbs are adducted and the fists clenched. The legs are generally rigid and extended; they may, however, be flexed. The head, trunk, and extremities are rarely all flexed together. The tongue is generally during this or in the next stage thrust between the teeth. Foam, saliva, and mucus drool from the mouth. The tonic rigidity may also entail a discharge of feces and of urine. The period of *tonic spasm* lasts from a few seconds to half a minute. During or immediately following it a tremor appears. Next comes the stadium of *clonic twitchings:* short impulses resulting in severe, generally symmetric movements, energetic twitchings of the muscles of the whole body. These increase in intensity, follow each other more rapidly, and may become so forcible as to produce injuries of the bones, joints, and tissues. Luxation of the humerus sometimes occurs. I saw a patient in whom every attack caused a bilateral luxation of the humerus.

The clonic spasms of the muscles of the jaw often cause the tongue to be bitten. The eyeballs also become affected by clonic movements. With the onset of the clonic twitchings air is forced into the lungs, the cyanosis disappears, and the respiration becomes accelerated and noisy. The pulse is generally more frequent than normal. The whole body may become covered with perspiration. The bowels and bladder are often evacuated at this stage, and ejaculations of semen may occur.

This stage lasts from one-half to five minutes. Towards the end of it the twitchings become more rare and are confined to fewer muscles. The attack rarely ceases with the cessation of the convulsions.

A *third stage* generally follows, in which the *coma* persists. The patient may open his eyes; he is, however, dazed, and often passes into a deep sleep, which may last for hours. This sleep is very similar to a normal one; the patient may be awakened from it, but is then confused and complains of headache. The breathing at the beginning may still be stertorous. Vomiting may follow, some epileptics vomiting after every attack.

In a typical case during the attack there is total amnesia, which occasionally has been present also a short time before the attack (Féré). Alzheimer speaks of a longer amnesia.

The following phenomena often accompany epileptic attacks:

The temperature is, as a rule, elevated, though only from 0.1° to 0.5° C. (Bonneville). A higher temperature is found only in the *status epilepticus*.

The reflexes are abolished during the attack (the conjunctival and corneal reflexes also, and particularly the pupillary light reflex), the knee-reflexes being also absent (if they can be tested at all), though they may be normal or exaggerated (Sternberg). This loss of the reflex excitability may last somewhat longer than the attack. Minute ruptures of the capillaries of the skin and mucous membranes, or circumscribed *hemorrhages* of the skin and conjunctiva, also occur. The urine passed during or just after an attack may contain some *albumin*. Polyuria may come on after the attack. The excretion of urea is also increased at times. This fact, though repeatedly observed, is combated by some. I observed it repeatedly in one of my cases.

The papilla is said to be pale at the beginning of an attack and to become hyperemic later (d'Abunds). Knies found the retinal arteries narrowed and also saw the ciliary muscles take part in the spasms. Instead of the post-epileptic sleep deafness and a dazed condition may occur, and increase to a noticeable mental disorder.

Motor weakness or paralysis, aphasia or stuttering rarely follow an attack. These transient paralyses are the result of exhaustion of the motor cortical centres (Todd, Jackson). The more pronounced these phenomena are, and the longer they last, the more probable it is that it is not a genuine but a symptomatic epilepsy. A *concentric contraction of the visual field* and *disorders of sensibility* are not rarely results of true epileptic attacks, as Thomsen and I have shown, and may persist for some time. Féré confirms this. An *erythematous flush* at certain places or upon a certain part of the skin may also follow an attack. Tremor, conjugate deviation, paralyses of ocular muscles, blindness, deafness, diarrhea, vomiting, polyuria, etc., have also been described as post-epileptic symptoms.

The attack need not follow the aura. In those cases in which it commences in an extremity, especially the hand, and consists of local paresthesia, or twitchings, the attack may be aborted by rapidly tying a string or cord around the extremity above the place where the aura arises. More rarely mere rubbing or pulling of the muscles has the same effect. One of my patients, in whom the spasm started in the hand, was able to abort the attack by suddenly and forcibly over-extending the hand. Two of my patients claimed that by an extraordinary effort of the will they were occasionally able to prevent an attack. This, at the most, occurs rarely, and in the absence of any control observations must be cautiously accepted. Roskam has lately made observations, however, which confirm this.

An atypical form of the epileptic attacks, in which the individual—as if driven by a hidden force—unconsciously runs some distance forward or perhaps backward before the real attack commences or without anything else occurring, is called *procursive epilepsy* or *walking epilepsy* (Bourneville, Ladame). It is said to occur mostly in degenerate individuals, particularly in morally perverse individuals. It may, however, alternate with the typical attacks, and we are not justified in assuming a special basis for this form. It forms a transition to those epileptic attacks in which the patient, driven by a secret desire for wandering, runs around for hours and days, or makes long journeys, yet conducts himself in a proper manner, although he is in an entirely altered mental condition. Afterwards the memory of these purposeless actions is absent or but fragmentary (Laségue, Le Grand du Saulle, Charcot).

In a few cases, the twitchings not only began on but were confined to one side of the body. The attacks resembled the cortical epileptic ones, and only the course and accompanying symptoms enabled one to differentiate them from Jacksonian epilepsy. In some cases the tonic or the clonic stage is absent, or the attacks consist only in the loss of consciousness (apoplectic form of Trousseau). These form a transition to the cases of minor epilepsy, or

Petit mal. The chief symptom, and often the only one, of these attacks is the loss of consciousness. This sets in suddenly, or more rarely is preceded by an aura. The unconsciousness occurs and generally disappears so suddenly—in from a few seconds to one-half a minute—that the individual does not fall, as a rule, and may resume his interrupted conversation or occupation, as if nothing had happened. Many patients of this kind are unaware of their attacks. Observation of them enables one, however, to recognize certain disturbances. The face generally becomes pale, rarely flushed, the eyes become fixed and staring, *slight clonic twitchings* occur, especially in the lips, tongue, and eyes. The tongue is generally not bitten, nor is the urine often passed involuntarily.

Instead of unconsciousness, a transient *vertigo* may form the attack, though the so-called epileptic vertigo, unless accompanied by some other symptom (momentary unconsciousness, transient twitchings, passage of urine), can hardly be diagnosed.

A condition of dream-like mental distraction often occurs in place of or following upon unconsciousness, in which the patients walk around, run away, undress, expose their genital organs, commence complicated and apparently premeditated movements, etc., without having any knowledge of what they are doing, or later remembrance thereof. These phenomena belong then to those remarkable psychic disturbances which form the *equivalents of the epileptic attack*,—*i.e.*, appear in place of the spasms, and show their relationship to them by alternating with them, and which, nevertheless, possess many characteristic peculiarities.

These *psychic equivalents* are not easily separated from the forms of the *post-epileptic mental disorders* which often follow the attacks. The same mental disorder which, when it occurs independently, is classed as an equivalent may follow an epileptic attack. Some authors even, as Magnan and Le Grand du Saulle, deny the existence of equivalents.

The many forms of epileptic insanity studied particularly by Morel, Falret, and Sammt have a number of common symptoms. We find generally a condition of complete *distraction*, in which the patient speaks incoherently, acts without motive or self-control, as in urinating on the floor, stealing, undressing before others, etc. After the attack, which lasts from a few minutes to an hour, all recollections of it are lost.

Epileptic delirium is characterized by a *paroxysmal maniacal excitement*, in which the patient, from an uncontrollable impulse, under the influence of hallucinations and delusions, is forced into committing wild acts, or becomes affected with a causeless anger, and may even commit deeds of the gravest and most horrible nature. It lasts for hours and days, and in a few cases for weeks. No recollection of this delirium, as a rule, remains afterwards.

These psychic equivalents generally come on suddenly, the patient being rarely warned by any premonitory signs. It is to a certain extent characteristic of them that all the attacks somewhat resemble each other. Complete unconsciousness does not occur in these conditions, only a peculiar disturbance of the consciousness; a dream-like, altered consciousness, in which apparently automatic, mechanical, and conscious acts seem to alternate with strange, purposeless ones (Siemerling).

Post-epileptic insanity is similar in many respects, as has been said before, to the psychic equivalents. It may occur under the picture of *epileptic delirium*, *stupor* with *distraction*, *terror* with *horrible hallucinations*, *apathy*, and *dementia*, and last a few hours to some weeks.

Sudden and severe outbreaks of perspiration, not due to any known cause, also occur as equivalents of epileptic attacks, with or without unconsciousness. They are, however, rare. A sudden sleep (*narcolepsy*) may also occur, and which may be regarded as an equivalent. In some cases the epileptic attack consists of an automatic repetition of certain senseless words or of verbigeration, etc. (Cheadle). This condition has been designated *prattling epilepsy* (*epilepsie marmottante*).

A general tremor combined with a clouding of the sensorium may likewise represent the attack. Certain forms of angina pectoris, spasm of the glottis, asthma, neuralgia, etc., have been looked upon as being abortive types of epilepsy, though it has by no means been proven. I am also not inclined to regard nocturnal enuresis as a frequent prodrome of epilepsy.

Reflex epilepsy to a certain extent commands a special position. It is caused particularly by injuries of the peripheral parts of the extremities (hand, foot). Reflex spasms are preceded by an aura which commences in the injured part of the body, and is felt as a paresthesia or twitching. It may remain in certain muscles or an extremity for a long time. The paresthesias and twitchings in succeeding attacks extend farther, until finally consciousness disappears. In these forms the attack is often followed by a paralysis of the extremity or of the side first involved in the spasm,—a paralysis which soon recedes. The scar is generally sensitive, and occasionally forms an *epileptogenous zone*,—*i.e.*, stimulation (rubbing, striking against it, etc.) will evoke a spasm, while it is just in these cases that binding the limb will inhibit an attack. I was able to satisfy myself in several cases that it is a *true epilepsy*, particularly by the pupillary rigidity which occurred and the other criteria of epilepsy. Some of the attacks caused by cranial injuries are cases of reflex epilepsy.

The *general state* of the epileptic is often normal. *Degenerative stigmata* are, however, frequent. Cranial malformations, errors of refraction, and congenital developmental anomalies are often observable.

The *intelligence* may be intact. Cæsar, Napoleon, etc., have often been referred to as examples; recent history, however, remains singularly free from them.

Depression, *mistrust*, and a propensity to become angry easily are often seen; *simple* dementia, and loss of intelligence, especially of the memory, also occur. Hypochondriasis is often associated with epilepsy.

The epilepsy of early childhood is often combined with imbecility or idiocy, and this mental weakness is co-ordinate with the epilepsy. But an originally normal intelligence may succumb to frequent attacks of epilepsy or to its long continuation. Minor epilepsy endangers the mind just as often as the major attacks. A long absence of the latter may induce a condition of indolence and mental weakness which will disappear upon the onset of a new attack.

Differential Diagnosis.—A diagnosis of epilepsy may be difficult from the fact that the attack is not seen by the physician, but merely described to him, and then only incompletely. A diagnosis of epilepsy should never be made from a single attack. In addition, the less the description accords with the classical type the more caution is necessary in making a diagnosis. This is particularly true of conditions of simple unconsciousness. An epileptic attack, for instance, may be confused with simple *syncope*. The latter, however, is due to anemia of the brain from fright, disturbance of the heart's action, etc., and, therefore, always follows some excitement or results from weakness of the heart's action. The epileptic attack in contrast occurs spontaneously and suddenly, or after a characteristic aura. In syncope the heart becomes weak or, at least, the pulse becomes small and weak. The loss of consciousness is, however, never so complete that the reflexes, particularly the pupillary reflexes, are absent. If convulsions, involuntary micturition, automatic movements, etc., accompany the unconsciousness, a diagnosis of epilepsy is almost always assured.

I had in one case great hesitation about making a diagnosis of epilepsy. It was the case of an otherwise healthy woman, having only some slight vasomotor disorders, who, during a banquet, at the onset of her menses, became totally unconscious, even evacuating feces and urine without exhibiting any motor symptom of irritation. After a quarter of an hour she awoke and thereafter remained well. I regarded it as being due to a cerebral anemia of sudden onset, caused by vasomotor disturbances; and, in fact, the woman, although years have elapsed, was never again troubled with a similar attack.

A diagnosis from *uremic*, *saturnine*, and other *toxic* attacks is not difficult. Ménière's vertigo may assume the picture of an epileptic attack. The aural symptoms are, however, always present, and lead to a correct diagnosis.

It is as difficult as it is important to differentiate *hysteric* from *epileptic* attacks. If the spasm occurs under the eyes of the physician, the most positive differential point is the condition of the *pupillary reflexes*, which is only and always lost in epileptic attacks. The sudden onset of the attack without any psychic excitement, the epileptic scream, the character of the twitchings (always completely involuntary), etc., speak for epilepsy. The arc de cercle, the salaams, the speech, the yelling and frenzy during an attack, the painful postures, the spasms of crying and laughing indicate an hysteric attack. The tongue is almost never bitten in hysteric attacks, while biting of the lips or snapping at surrounding persons is especially characteristic of them. I observed, however, one hysteric person bite her tongue in an attack. Hysteric persons also rarely hurt themselves much in falling, as do epileptics. Involuntary micturition, and especially defecation and pollutions during an attack, speak for epilepsy. Charcot claimed that involuntary micturition during an attack may also occur in hysteria. This author also called attention to the fact that hysteric attacks are especially common in the evening, epileptic ones in the night or early morning (three to six A.M.). Deep sleep after an attack indicates its epileptic nature.

If the patient be seen between attacks, all these points must be inquired into. If scars are found on the tongue, acquired during the attack, it indicates epilepsy. If the attacks are of long duration, the mental condition must be investigated. Intact intelligence, grimaces, and a sharp intellect speak for hysteria; a stupid expression or dementia indicates epilepsy. These criteria should not be considered absolute.

I remember walking through the convulsion ward of the Charité in company with the physician-in-charge, and attempting to diagnose the character of the attacks from the facies, and succeeding in all but a few.

The length of the attacks also differs in these two diseases. An epileptic attack, excluding the sleep following the paroxysm, lasts only a few minutes (one to ten). In attacks of from one-fourth to one-half hour hysteria or an organic cerebral disease is present. In doubtful cases, hypnosis or other suggestive influences may be tried to institute an attack. The success of such a manœuvre speaks for hysteria. The following is also of importance: pressure upon a certain place, for instance, ovarian pressure, will in hysteric cases repress or influence the convulsions.

Not only mixed forms of hysteria and epilepsy, but also intermediary conditions exist. In the psychic forms of epilepsy the hallucinatory delirium and the conditions of distraction offer difficulties, being also found in hysteria. The degree to which the brain is befogged, the force with which all acts are done, and the purposeless onset of the attacks characterize the hallucinatory delirium of epilepsy.

The convulsions occurring in children as a symptom of fever are not epilepsy. Those spasms occurring in individuals who are troubled with intestinal worms cannot always be distinguished from true epilepsy. It is claimed that in some spasms caused by tænia the attack comes on gradually and the phases last longer than those of true epilepsy. The best method of differentiating all these so-called reflex spasms is by the results of treatment.

Nocturnal epilepsy may for a long time be unrecognized, especially when the patient sleeps alone. Biting of the tongue, blood on the pillows, micturition during sleep, hemorrhages on the skin and conjunctiva, contusions of unknown origin, dull headache, and a dazed condition and depression in the morning, serve to detect it. Irregularity of the respiration, gurgling and gurgling sounds, etc., indicate an attack in the night.

Genuine epilepsy cannot always be distinguished from *symptomatic* epilepsy. If the epileptic attack is merely a sign of increased intracranial pressure, as in many cases of cerebral tumor, other symptoms will generally be found to indicate this. It is important in every case of epilepsy to seek for signs of an organic cerebral disease, and above all else to make an ophthalmoscopic examination. It should, however, be remembered that epileptic attacks may precede by years the development of a brain tumor, so long indeed that one could hardly speak of a latency lasting that number of years.

If the attack is of a *cortico-epileptic* character, it is very probable that genuine epilepsy is not present. In some cases of the latter the convulsions do confine themselves to one side of the body; they do not then show the regularity in development and course, or the progressive character of a cortical epilepsy, nor are the other symptoms of the latter present. It should be regarded as a rule that the convulsions in true epilepsy rapidly become general. The border between partial and general epilepsy is, however, not always sharply defined. It should not be forgotten that cranial injuries tend to excite, through the scars which they leave behind, a reflex epilepsy, which is often wrongly referred to cerebral focal disease.

Attacks of spasm are not rarely observed at the beginning and during the course of *dementia paralytica*. They are generally unilateral spasms without loss of consciousness. The typical attacks do not belong to the common phenomena of this disease. The other signs of this disease aid in making a diagnosis.

Epilepsy in later life is generally symptomatic, and is due to a cerebral tumor, cerebral syphilis, general paresis, chronic nephritis, cysticercus cerebri, arteriosclerosis, etc.

It is more difficult than in adults to assign causes for and differentiate the spasms occurring in early childhood. The spasms of dentition should not, without further proof, be regarded as epileptic. I regard it just as unjustifiable to look upon the so-called salaam spasms as a species of epilepsy as does Féré.

In those rare cases in which an infantile cerebral palsy does not leave any motor disturbance, the accompanying epilepsy is characterized by the unilateral onset of the twitchings; close examination will also reveal a tendency, though, perhaps, slight, to accessory (involuntary) movements and athetosis.

In *simulation* of epileptic attacks, pupillary rigidity, pallor at the beginning of the attack, cyanosis, biting of the tongue, and the post-epileptic daze and mental distraction, etc., are absent. Mairet lays stress upon the urinary examination; the increased phosphates and nitrogenous elements, the decrease in toxicity resulting from the attack, and the "hypotoxicity" of the urine in the intervals (discussed lately so much in French literature) are characteristic signs of epilepsy. Further investigations must, however, determine the value and significance of these moments.

Course and Prognosis.—The frequency of the attacks is more variable than is their type. One patient will have daily seizures; another, one seizure in a year. As a rule, we find one or two a month. In petit mal there may be twenty or more attacks in a day.

If the major attacks follow each other, one after another, without the patient becoming conscious, we speak of a *status epilepticus*. This is a dangerous condition. The temperature increases at each attack and may reach 41.5° C. (107° F.). In one case, ending in death, I found 42° C. (107.4° F.); while Bourneville found a post-mortem increase to 44° C. (111° F.). The pulse is small and frequent. The attack may last a few days and produce marked exhaustion or even death.

Many epileptics have only major attacks; others, only minor ones, and some suffer from both.

The attacks occur more often by day than by night. If they occur more often or only at night, they may be unnoticed for a long time. In women they often occur at or about the menstrual time. During pregnancy they may disappear, though this is not a rule. Acute pyrexial diseases, also injuries, operations, or chronic suppuration, may inhibit the spasms.

The individual attacks are generally ushered in without any known cause. The patient occasionally blames it on indigestion, overwork, coitus, etc. In hospital treatment it has often been observed that the attacks increase after each leave of absence. Psychic influences, as terror, rage, etc., act in this way, and for this reason we are not justified in doubting the existence of epilepsy. In a child suffering from infantile spastic hemiplegia and epilepsy I was able to produce an epileptic attack by frightening it. The parents called my attention to this fact, and claimed to have seen a second or counter-fright restrain the onset of an attack.

The disease is not very dangerous to life, though not a small percentage of epileptics die early. Life is threatened, particularly in the status epilepticus. Of those who arrive at this condition about one-half die. Others die as a result of wounds or injuries which they acquire in falling; for instance, a fall into a fire. Suffocation may also produce death, particularly in those who lie upon their face during an attack. Asphyxia or cardiac rupture occurring in an attack rarely leads to death.

Complete recovery, unfortunately, is a rare occurrence, though the number is greater than is generally supposed. I have often discovered that the relatives of an individual who consulted me on account of epilepsy or some other nervous disease were afflicted with spasms in their youth, which, according to their description, must have been epileptic, and who five and twenty years afterwards were, nevertheless, entirely free from attacks. Persons whom I have treated in their mature years for neurasthenia, hemicrania, and other diseases of the nervous system, have sometimes told me that in their childhood, until their fifth to eighth year, they had epileptic spasms. This fact seems to me important, because in cases of epilepsy which come under our care we may only speak of recovery when our observation has lasted for at least the greater part of a decade.

Cases accompanied by an acquired or inherited mental weakness have a bad prognosis. The longer the disease has lasted the less chance is there for recovery.

The prognosis does not directly depend upon the severity of individual attacks. It appears to me that the prognosis in petit mal in regard to ultimate recovery is worse than in major attacks. Alcoholic epilepsy is more often cured than genuine epilepsy. Syphilitic forms are also occasionally amenable to treatment. Reflex epilepsy is also frequently cured.

Those forms due to cranial injury offer chances for cure only when they are of a cortical or reflex type.

Pathologic Anatomy and Pathogenesis.—Post-mortem examinations, as a rule, are negative. At least no alterations are found which can be regarded as the cause of the disease.

Thickenings of the cranial bones and of the meninges are such irregular conditions as to have no importance. The *sclerosis* of the *cornu ammonis*, which Meynert referred to, and the importance of which Sommer and lately Bratz, who found a hypoplasia of the cornu ammonis, mention, may be a developmental anomaly, and merely a "stigma hereditatis."

Whether the abnormal narrowness of the cerebral arteries and of the aorta, which is described now and then, plays any part in the pathogenesis is questionable.

Lately, histologic examinations of the cerebral cortex have produced remarkable results. After Bevan Lewis, Buchholz, and others found conditions which seemed to indicate a disappearance of nerve-cells of the cortex, Chaslin and others made more exact observations on the brains of epileptics. He found an hypertrophy of the glia fibres in the cortex, —that is, a form of *sclerosis* or gliosis, even noticeable macroscopically. Bleuer has confirmed this, and has proven particularly the occurrence of an hypertrophy of the external glia layer. It is a question still unanswered as to whether it is a cause of epilepsy or a result of the attacks, as Marinesco and others claim.

Though pathologic anatomy has not given us anything definite as to the seat of the disease, the experimental observations of Fritsch and Hitzig, Unverricht, Francois and Frank, and Pitres, etc., have shown that epileptic attacks may be excited from the motor zone of the cortex, and that by the extirpation of certain cortical areas the convulsions in the corresponding muscular groups may be silenced; we know, further, that diseases of the motor zone, which are accessible to the pathologic anatomist, lead to the same phenomena. These facts have been the best refutal of the opinion formerly held by many, particularly by Kussmaul and Nothnagel, that the medulla oblongata and the pons were the seats of the disease.

Although these convulsions differ in their unilateral onset from true epilepsy, this is due to the fact that the irritation, whether due to an artificial cause or to this disease, begins in the motor zone of one hemisphere. If we would assume as a cause of epilepsy a disease of the motor zones of both sides, this difference between the symptoms would be to a great extent explained. The psychic disturbances, which follow or replace the attack, and also the mental weakness which frequently comes on during the disease, indicate particularly that the cerebral cortex is the seat of this disease.

The fact that in diseases which produce a complete break of conduction in the motor tract of the inner capsule the paralyzed half of the body may be free from convulsions, also indicates its cortical origin. Lately, however, some authors (Binswanger and others) have favored the idea that lesions of subcortical centres, particularly of the medulla oblongata, help to evoke the convulsions.

The epileptic attack is looked upon as being due to a discharge of accumulated irritation (Hughlings Jackson). Schroeder van der Kolk compared it to a Leyden jar.

Some authors are inclined to the view that an encephalitis of the motor zone, which had existed in childhood, may cause the same alterations as those which are the basis of epilepsy.

In recent years the attempt has been made to refer the epileptic attack to an autointoxication. This opinion, which has been particularly advocated by Voisin, Féré, Haig, Bouchard, Krainski, and others, is based upon the fact that the urine after an attack is richer in toxic products, and that by introducing it into the blood of animals used for experimental purposes it shows that it is more poisonous than that passed before the attacks and in the intervals. At such times it is hypotoxic, after an attack hypertoxic. They deduce from this that the retention of toxic material in the blood produces an autointoxication which expresses itself by epileptic attacks (and by gastric disorders, etc.). Definite chemical bodies, as uric acid (Haig), ammonium carbonate (Krainski), have been named as being the toxic materials. Voisin insists that a congenital disposition is a most important etiologic factor, while the autointoxication is the exciting moment. A congenitally produced abnormal condition of the nervous system probably produces a temporary disturbance of the secreting power of the kidney, and this causes a retention of toxic material in the blood, etc.

At any rate, the observations given above deserve further attention, though we are by no means justified in regarding the autointoxication theory as being firmly established.

In reflex epilepsy it is the irritation of a scar which acts upon the motor zone and evokes in it "the epileptic alteration."

Treatment.—The chief indication in the treatment is to undertake a thorough examination of the body and to remove, if possible, all exciting causes. Even those factors whose genetic relationship to epilepsy is still doubtful, as tænia, diseases of the nasal mucous membrane, gastric and intestinal diseases, etc., must be taken into consideration. For before we can conscientiously undertake any symptomatic treatment we should endeavor to stop up the source of the trouble, although it is rarely possible. It should not be regarded as too trivial to prescribe a *tape-worm cure*, to remove a *nasal polyp*, or an *aural polyp*, or to combat an *obstipation*. No harm can be done, and the possibility of a cure thereby is not excluded.

Satisfactory observations of the cure of epilepsy by the extraction of a tooth, polyp operations, removal of foreign bodies from the ear, nose, etc., are, it is true, very few.

Diseases of the sexual apparatus should also be attended to, although it is very doubtful that they ever produce epilepsy. *Injuries* should especially be sought after. If the aura arises from any definite part of the body, it is advisable to search for *scars* or other signs of a previous injury. If it can be proven that reflex epilepsy is present, *extirpation of the cicatrix* is indicated. This treatment is often enough unsuccessful or only of temporary benefit (probably because the cortex has been chronically altered), but that should not make us hesitate to enter upon the rational way to a cure.

If the disease can be referred to alcohol poisoning, the abstinence from alcohol must be enforced, preferably in an institution. A symptomatic treatment is generally required in addition. If syphilis is suspected,—it may have been inherited as well as acquired,—an iodide and mercury treatment is indicated. It is desirable to combine the bromides with the iodide of potassium.

In respect to the nutrition, all *irritating* food or drink must be forbidden. Condiments, dressings, alcoholic drinks, and strong coffee and tea must be forbidden. Cases have been observed in which an epilepsy cured for a long time broke out anew after a drinking bout (Maisonneuve). In addition, only those eatables must be taken which do not easily cause indigestion; the stomach must not be overloaded. The diet should be a mixed one. Meat should only be taken in moderate quantities.

In nocturnal epilepsy the patient should take only a light supper, and that very early in the evening.

Adequate exercise in the open air is desirable, though the patient must never be permitted to exhaust himself. Mental overstrain is even more inadvisable. If the patient has still the choosing of a vocation before him, one should be sought out which offers no danger to an epileptic. Work upon a new building, near or upon water, by an oven, etc., are not adapted to epileptics. If the attacks recur at only rare intervals, and if the intelligence is intact, the choice of a calling requiring principally mental work is not permissible. The more it requires an association with the outer world, the less adapted is it to an epileptic. Manual labor which does not place the patient in too much danger is a very good occupation. Farming is excellent. It is always advisable, however, to see that epileptics are under observation as much as possible, as harm may befall them otherwise.

In those cases of epilepsy in which *temporary insanity* replaced or followed the attack, the patient should be placed in an institution.

The *mentally weak epileptic* should also be cared for in an asylum. Those who are troubled with frequent major attacks are also better cared for in institutions.

State asylums or colonies conducted under medical supervision and entirely devoted to the care, treatment, education, and occupation of epileptics, are especially advisable. The city of Berlin has a special institution for epileptics at Wuhlgarten. Colonies modelled after the German ones have been established in a few States of the United States. There are also a number of private institutions in which much attention is paid to the treatment of epileptics.

Climatic treatment is, so far as my experience goes, not a curative factor; but a stay in the country and forests may be beneficial by favorably influencing the nervous system and by removing many injurious moments. A mild *cold-water treatment* may be used in every case; sea-baths, of course, are dangerous. I have not observed any particular benefit from high altitudes.

The results of electric treatment are very doubtful. Galvanization of the brain, also of the sympathetic, may be tried.

Treatment of the Patient in the Epileptic Attack.—If the aura originates in any single extremity, the attempt may be made to cut short the attack by tightly constricting it before the aura has extended farther. This succeeds in some individuals. They may be instructed to carry a small girdle with holes in it—similar to the old leather garter—and use it as a ligature. Forcible flexion, extension, or pulling of a limb may also inhibit an attack (Bravais). On other occasions, energetic peripheral stimulation sufficed. Artificial repression of an attack is, however, not always advisable; it often leaves behind a condition of depression and irritability, combined with headache and vertigo, so that many prefer the attack. Inhalation of *amyl nitrite* (a few drops on a handkerchief), the administration of chloral hydrate in hypnotic doses, or the ingestion of a teaspoonful of salt may, in some cases, stop the attack. If the attack breaks out, notwithstanding these remedies, our duty is to see that the patient is protected from injuring himself, that the clothes are loosened around his neck to aid the flow of blood from the head, and that a piece of cork or gum is placed between the teeth of those who are in danger of biting their tongues.

It is not advisable to awaken a patient from his post-epileptic coma or sleep, as headache and psychic depression are bound to be produced. In *status epilepticus* prescribe chloral hydrate in doses of forty-five to sixty grains, per clysma. Hypodermic injections of morphine are not very efficacious. Wildermuth praises amylen hydrate in doses of five to eight grains. If these remedies do not act, *bloodletting* is in place.

Purgatives, diuretics, lithium carbonate, and intestinal antiseptics are given by those who believe in the autointoxication theory.

In most cases, we find it necessary to use drugs in our treatment. Whenever the attacks occur only at long intervals, perhaps once in a year or in the course of several years, I do not regard it as justifiable or even rational to give any drugs. In all other cases, whenever the causal indication has been attended to, it is necessary to prescribe some drug which will relieve the convulsions or at least lessen their number and intensity.

The bromide salts are the most serviceable of all the drugs at our command. Authors differ exceedingly as to the manner of using them and the proper dosage. It is advisable to commence with small doses—forty-five to seventy-five grains a day in adults—and gradually increase the dosage until the amount required to repress the convulsions is ascertained. It may be necessary to increase the daily dose to one hundred and eighty or two hundred grains. Smaller doses must be administered to children, though from the fourth year fifteen to sixty grains a day may be given.

According to Féré, success is only secured when the bromides produce sleepiness, a desire to sleep, etc., and when the absent pharyngeal reflex betokens that they have taken effect (Séquin). He gives continued large doses and persists in their administration, even though attacks have long ceased.

It is the rule to have the patient use the drug for a long time—a year or several years—and only to stop its use when a condition of exhaustion combined with hebetude, gastric disorders, and an impaired cardiac action become noticeable, showing that the patient is suffering from bromism. Poisoning from the bromides rarely expresses itself by conditions of exaltation. Bromide acne rarely becomes so severe as to demand the cessation of the treatment. Only when the attacks have ceased for about a year should the drug be discontinued. It is also necessary to dilute well the bromide used. Some prefer the potassium, others the sodium, others the ammonium salt, and some a mixture of the three. A slightly alkaline water may also be used. The bromide may also be dissolved in a cold infusion of valerian root. It is not recommendable to administer the bromide in too many doses, two or three being plenty. If the attack occurs at a definite time of the day or night, it is advisable to give the *entire dose* from four to six hours before the usual time of the seizure. Some administer the drug in correspondingly large doses only every few days.

In some cases it is possible to keep off the attacks entirely with the bromides; in others, however (and these are the most frequent), they only become rarer; in one case, for instance, in which the attacks had always come on every fourteen to twenty-eight days, the use of about forty-five to sixty grains of the bromides stopped them for five years. The remedy often fails or is not borne well, and it is necessary to use another drug.

Atropin is the next best drug to use for epileptic attacks. I have seen good results from its use in several cases where the bromide had entirely failed. This drug also is to be given at first in small doses, using one-third to one-half a milligram several times daily in adults and correspondingly smaller doses in children, to be gradually increased. I have been able to use this drug continuously for years with frequent interruptions. *Belladonna* is also occasionally efficacious.

An old formula for a pill was extract of belladonna and belladonna leaves of each fifteen grains, extract of liquorice sufficient to make one hundred pills. S. At first one or two, increasing to from four to six pills a day.

Potassium bromide may be given in combination with extract of belladonna, the latter in doses of from one to two centigrams. H. Jackson advised the use of the latter drug in increasing doses. *Radix artemisiæ* and the zinc salts, particularly the oxide and valerianate of zinc, have only an historic interest. With this disease, however, we are often desperate, and in groping around may fall back upon these older remedies. The cocculus fruit and sodium nitrite may be mentioned for the same reason.

Nitroglycerin, given in alcoholic solution in doses of from one-tenth to five-tenths of a milligram, need hardly be mentioned.

Of the newer preparations of bromine, *ethylen bromide* seems to be the most efficacious.

℞ Ethylen bromide 5.0 ad emulsio oleos. 100.0, ol. menth. pip. gtt. 2.—Adults should take thirty drops in half a glass of sweetened water two or three times a day, increasing ten drops every third day until seventy drops, or a teaspoonful, are being taken. Children of from eight to ten years should commence with ten to twenty drops. The drug may also be given in gelatine capsules, each one holding three drops combined with six drops of the oil of sweet almonds, from two to four to be taken daily.

Satisfactory observations of the curative effect of this drug have not been carried out long enough to say anything certain about it.

Strontium bromide, camphor monobromate, calcium bromide, gold bromide, zinc bromide, and bromalin have been recommended.

Amylen hydrate in watery solution (one to ten) has been given in doses of from two to four grams. Combinations of the bromides with antipyrin, *opium*, digitalis, and adonis vernalis, have been recommended, also with chloral hydrate in particularly severe cases.

Borax has been recently offered as a substitute for the bromides. It should be given in doses of from ten to fifteen grains, and gradually increased to one dram after meals. After long use it may produce an eczema and a conjunctivitis, which are to be treated with arsenic, as is the bromide acne.

An epilepsy of supposedly malarial origin should be treated with quinine.

The *combination of an opium treatment with the bromides* (Flechsig) has lately been recommended. Doses of opium (the extract) of from one to fifteen grains a day are given, beginning with the first and gradually increasing the dose to the latter quantity. After six weeks the opium is withdrawn, and large doses of the bromides (about two drams daily) are given. After two months the dose of the bromide is decreased to thirty grains daily.

On the whole, the results of this treatment are very doubtful, and, in addition, it is not harmless, does not suit those of weakened constitutions, and demands careful watching, and is therefore probably only practicable in a hospital.

If epilepsy develops in those who are troubled with cephalic congestion, and in whom the signs of cerebral hyperemia are present in the intervals also, it is justifiable occasionally to undertake bloodletting.

Operative treatment is demanded in almost all cases of *reflex epilepsy* and of *traumatic epilepsy*. The observation that every injury is able to keep back the convulsions for a certain length of time has led to the artificial production and maintenance of suppuration. I saw Westphal in the last years of his life still apply a seton to the nuchal region in some cases of epilepsy. This method of treatment is now only recommended by a few physicians. Féré applies the cautery to the head. In those cases where an aura arising from a definite part of the body ushers in the attack of true or of partial epilepsy, the attempt may be made to abort the seizure by a blister continuously applied to the limb above this place. Bravais, Hirt, and Buzzard have had good results from this method.

Reflex epilepsy demands the excision of cicatrices, their release from bones, the freeing of a nerve from a callus, the removal of foreign bodies, of a growth, etc. If these measures are unsuccessful, the nerves in question may be stretched. This in one of my cases, in which a cicatrix was not found upon the extremity from which the aura started, led to recovery. Unfortunately, in only a small percentage of cases can a permanent result be counted upon. The operative treatment should be supported by other measures (bromides, etc.).

Epilepsy due to cranial injuries often demands surgical interference.

Cortical lesions (adherence of the upper cortical surface to the meninges, scars, cysts, long splinters, and the like) in the motor zone are generally present, or it is the pressure to which the cortex is subjected by a depressed bone which causes the irritation. Notwithstanding, the attacks are not always of the character of cortical epilepsy. It should be remembered that injuries which do not harm the bony cranium, on account of the elasticity of the cranium, may affect the cortex.

If a cranial lesion has preceded, trephining may be beneficial (1) when the attacks resemble those of cortical epilepsy, and (2) when the cicatrix is over the motor zone. According to Horsley and others, however, not only the bones, the cicatricial meningeal tissue, the cysts, etc., must be extirpated, but also the cortical centre from which the irritation arises. Others have advised against this procedure, though Sachs and Gerster have lately advocated it again. Some have gone farther, and even where no cranial trauma could be determined upon, and where no scar could be found, have operated in a similar manner, whenever the attacks resembled those of Jacksonian epilepsy. The centre in question was in such cases determined upon by electric stimulation. It should be remembered that, notwithstanding the normal appearance of the cortex in cases of partial epilepsy, the microscopic examination may reveal distinct alterations (Coën, v. Gieson, Collins, myself).

In general, the results of operative treatment in traumatic epilepsy are not very encouraging. One fact has particularly been shown,—namely, that in most cases the effect of the operation is but temporary (v. Bergmann, Raymond, and others). Sachs and Gerster emphasize the fact that results are more liable to be favorable in recent cases, in which the injury occurred at the most from one to three years before. In one case (Larrey) the extraction of a bony splinter thirty years after an accident was efficacious. More exact indications cannot be set up, though, from the facts detailed above, an operation should most probably be undertaken only as a last resort, and then only when the epilepsy is very severe. Alcoholism, according to the experience of Jolly and others, might be a counter-indication.

V. Bergmann does not believe that operative procedures are advisable in traumatic cases whose course resembles that of true epilepsy. It is also not justifiable in cases of the psychic equivalents of epilepsy, even when a cranial injury has preceded.

Ligating the carotid and vertebral arteries need hardly be mentioned as methods of treatment. The extirpation of the chief sympathetic ganglion, undertaken by Alexander, is merely mentioned as a curiosity.

ECLAMPSIA.

I. INFANTILE ECLAMPSIA.

General convulsions are frequently observed in early childhood. Although generally corresponding to the type of epileptic attacks, they occur under conditions, and present differences in course and prognosis, which make it necessary to separate them from epilepsy. In this sense it is justifiable to group these general convulsions of early childhood under the designation *infantile eclampsia* in order to study them more closely.

Conditions to which adults do not react with spasms can produce in children general convulsions with or without unconsciousness. This is referred to an *increase of the general reflex excitability*. It seems to me, however, that an *incomplete development of the inhibitory centres* is the factor which renders possible the onset of spasms from the most varied causes. A certain analogy exists, then, between a child's brain and that of an hysteric patient.

It is not uncommon for the acute infectious diseases to be ushered in by convulsions in children. *Gastric and intestinal diseases* are often (according to Morselli, generally) the causes of infantile eclampsia. Gastro-intestinal catarrh, intestinal worms, even simple gastric disturbances (overloading of the stomach), *profuse diarrhea, dentition*, etc., may all cause eclampsia in children. *Rhachitis*, inguinal hernia, fright, and injuries have also been given as etiologic factors. The causes are, therefore, *toxic, reflex*, and *psychic* moments. In many cases a cause cannot be discovered. In what way rhachitis leads to eclampsia is not known. The general nutritional disturbance, the effect of chemical bodies, the softness of the cranial bones—all these factors may be in play.

The attacks are very similar to epileptic ones, but have a tendency to occur in series. General tonus alternating repeatedly during an attack with clonic twitchings also occurs. Mild convulsions of short duration, accompanied by screaming and intact consciousness, are also observed. Unrest, irritability, and partial spasms may be premonitory symptoms. Attempts to classify the different types of eclampsia are futile. In some cases the convulsions cease when the cause is relieved, the disease is confined to a few seizures, or they last for days or weeks and then disappear forever. In others they extend over a period of months or a year or longer before they pass away altogether. If the individuals are observed further, we find that many are affected with *epilpesy* or *hysteria*[1] in later life.

---

[1] The observation given by me in the first edition of this work, that eclampsia is often the first sign and prodrome of hysteria, has recently been confirmed by many, as, for instance, by Bruns and Fürstner.

It is definitely settled that the attacks of eclampsia often denote an *epilepsy* appearing in early childhood; that *hysteric attacks* may also occur at this period I know from personal experience. It should also be recognized that infantile eclampsia in many, perhaps most of the cases, is merely a peculiar *temporary* trouble, which may endanger life, but which, when over, is without any import for the future.

Palpable diseases of the infantile brain likewise often evoke spasms. If we exclude meningitis, which can be recognized, as a rule, earlier in its course by other symptoms, the organic cerebral diseases of childhood generally express themselves by unilateral convulsions.

Prognosis.—The prognosis is favorable in respect to single attacks, and becomes graver if they persist long and if they occur often. Severe and rapidly repeated attacks are particularly dangerous in weak children. The fear that it is an epilepsy is probably true if the eclampsia persists after removal of the poison and when a cause cannot be discovered. If consciousness is not lost, and if in general it resembles an hysteric attack, the chances for the preservation of life are good. Eclampsia must always be carefully differentiated from convulsions produced by organic cerebral disease.

Treatment.—The cause must first be removed, if possible. A gastric catarrh or a simple indigestion must be cured, thread-worms removed, etc. In a case of difficult dentition the gums must be incised. Rhachitis should be treated in the ordinary way. *Bromides* should be given against the attack itself, regulating the dose according to the age of the patient. Chloral hydrate, in small doses, or chloroform inhalations are advisable when the bromides do not act and the attacks seem dangerous by reason of their severity or frequency. Warm baths and wet packs are also good procedures, while cold douches are particularly serviceable in hysteric convulsions, as is also a mustard plaster on the nucha or extremities.

II. PARTURIENT ECLAMPSIA (ECLAMPSIA OF CHILDBIRTH).

These spasms occur in the latter part of pregnancy, during delivery, or after it. Young primiparæ are especially liable to them. The attacks resemble those of epilepsy; they have, however, a greater tendency to occur in series.

Eclampsia of pregnancy is due to *kidney disease*. This is the almost unanimous opinion of authors. It is generally an acute, more rarely a chronic, nephritis, perhaps also a compression of the ureters, which causes it. *Albuminuria*, edema, sometimes amaurosis, and retinitis are found present in these cases and reveal the basic disease. The attacks are then very similar to *uremic* ones.

It is probable that an increased predisposition of the nervous system, due to the gravid uterus or to the puerperium, is a factor. Recent investigations appear to show that an *infection* from the uterus, a specific bacillus, is the cause of the eclampsia gravidarum (Neumann, Gerdes). The view is not yet sufficiently supported by evidence to be accepted, and less so the opinion that the disease is due to the absorption of products of metabolism from the fetus.

The disease is a grave one and endangers life. Twenty-five per cent. of the cases die (Olshausen) in coma, in an apoplectic attack, or from sepsis. Fat emboli in the lungs have repeatedly been observed (Virchow). Delivery of the child renders the prognosis more favorable.

Treatment.—Two indications are to be carried out: (1) to hasten birth; (2) to lessen the excitability of the brain. Views differ as to the best method in the first indication, but the latest tendency is in opposition to a too forcible hastening of delivery. Some, however, would hasten delivery by incisions in the vagina and cervix even when the latter is still closed (Dührssen) or even by undertaking a Cæsarean section. *Continued chloroform narcosis* is used with success for the attacks, also *morphine* and *chloral hydrate* in large doses. *Venesection* and diaphoresis are good procedures, and active purgation is also in place.

LOCALIZED MUSCULAR SPASMS.

FACIAL SPASM (SPASMUS FACIALIS; CONVULSIVE TIC).

The facial muscles are the most frequent seat of spasms. This is most probably due to the intimate relationship between mental acts and movements of the facial muscles, and also to the rapid reflex actions which occur in the face. Both moments play a prominent part in the causation of these spasms. Painful diseases of the conjunctiva and cornea, carious teeth, and every *disease of the sensory trigeminal* may produce a facial spasm through *reflex action*. It is, therefore, not uncommon for tic douloureux and convulsive tic to be found together.

It is not settled whether disease of other parts of the body can evoke facial spasm in this way. Such an effect has been ascribed to *sexual diseases*, especially uterine disorders, and therapeutic results have been announced which would seem to indicate it. Gowers observed a facial spasm occur during pregnancy and disappear after the birth. Bernhardt noticed similar connections between pregnancy and facial spasms.

In many cases the facial spasm has a *psychogenic* origin, resulting from some emotional shock or protracted excitement. I observed several individuals become affected with a facial spasm during an earthquake.

This disease generally attacks only the *neuropathic*. I saw a contraction of the orbicularis palpebrarum, which had been voluntarily produced to counteract a squint or was of reflex origin, pass into a convulsive tic. It is rarely directly inherited. A direct *stimulation of the nerve-trunk* is only rarely a cause. A few observations exist which show that a compression of the nerve at the base of the brain (tumor, aneurism) may be the cause. It is possible, however, in these cases also, that a compression of a sensory nerve, which had been overlooked, may have produced it *reflexly*. At least, I saw a facial spasm come on in a case of extracranial tumor of the anterior cranial fossa which had injured the first trigeminal branch.

A form of partial facial spasm has been described as an occupation neurosis in watchmakers (T. Cohn). It is certain that overuse of the facial muscles, especially of the orbicularis, in such occupations increases the predisposition to convulsive tic.

The contracture which results from facial paralysis is not a facial spasm in the narrower sense of the word. But clonic twitchings may occur in the previously paralyzed muscles, perhaps as a result of minute alterations of the nucleus, which keep it constantly stimulated (Hitzig and others). These twitchings may also pass to the other side.

In contrast, otologists report that a facial palsy of otitic origin is occasionally preceded by a period of clonic twitchings.

It is rare for an *organic disease of the cortex* in the region of the facial centre to produce a muscular spasm limited to the region innervated by this nerve; we observe in these cases, generally, the phenomena of cortical epilepsy, which, it is true, commence in the facial muscles, but soon extend to the arm, and finally over the entire side.

Facial spasms which follow upon *cephalic injuries* may be due to an organic disease, but they are so often phenomena of the traumatic neuroses that in this genesis also functional disturbances are mostly in play.

Those diseases in which the facial spasm is merely a symptom, as epileptic and hysteric spasms, chorea, etc., will not be discussed here.

In most cases the facial spasm has no anatomic basis, and, as has been assumed, *minute* (molecular?) alterations in the nucleus or cortical centre are probably the cause of the irritation which reveals itself by the spasms.

Symptoms.—Facial spasm is generally unilateral. It involves the entire facial region—though generally the digastric and stylohyoid, and often the orbicularis oris also, are spared—or only single muscles are affected. The orbicularis palpebrarum shows a special tendency for involvement, and it is generally a bilateral affection in such cases. The zygomaticus may also be affected by itself; also the levator alæ nasi et labii superioris, more rarely the orbicularis oris and the muscles of the chin. A spasm involving only the chin muscles (geniospasm) has been described by Massaro as an hereditary disease. I have only occasionally seen a convulsive tic confined to the nasal muscles, and in but few cases one limited to the frontal muscle. In a case of Stembo, in which the occipitofrontalis muscle was involved by itself, hysteria was found present. It is exceptional for the spasm to confine itself to the external auricular muscles (Romberg). Bernhardt observed twitchings in the occipital muscle and in the retrahens auriculæ. The diffuse spasm extends to the platysma, and often to the auricular musculature.

The spasm is generally a *clonic* one, though clonic twitchings may be combined with *tonic* contractions and alternate with them. In tonic contractions the palpebral fissure narrows even to closure of the lids, the forehead is corrugated, and the nose and mouth are drawn toward the side of the spasm. The face presents numerous folds.

The clonic twitchings are often so slight as to be detected only on close examination; at other times so pronounced as to cause lively grimaces. They are short, lightning-like twitchings, which rapidly repeat themselves or only run over the muscle once. *Paroxysms* generally occur in which the contractions follow each other rapidly, while a complete cessation, or, at the most, a few weak twitchings occur in the intervals between the seizures.

They are decreased by mental and physical rest and are increased by *emotional* outbursts, *exertion*, especially in *chewing* or *speaking*, and by the stimulation of cold air. They may also be evoked by gently tapping the cheek, or by suddenly inspecting the eye, or by rapidly running the fingers over the facial skin. Some patients are able to inhibit the spasm volitionally.

In some cases, especially in *blepharospasm*, *pressure-points* are found in the trigeminal region (v. Graefe), at the exit of the supraorbital nerve, etc., slight pressure upon which inhibits the spasms. It is often, however, merely a suggestive influence. Such pressure-points at distant places—for example, upon the vertebræ—are rarely found. The spasm of the lid is generally a tonic one (blepharospasm), though a clonic form (blepharoclonus, nictitatio) is not infrequent. The first may be so persistent as to keep the eyes closed for weeks and months. The observation of Graefe, that in children a temporary blindness accompanies a blepharospasm which has been present for any length of time, is very interesting. Its nature is still undecided, notwithstanding numerous investigations (Silex and others).

The subjective disorders of this affection may be slight or severe. They depend upon the intensity of the spasm and whether the orbicularis palpebrarum is involved. Some individuals are remarkably tolerant to these spasms ; the clonic form is repeatedly observed in laborers without their being aware of it.

Voluntary facial movements are not impaired, though the spasm may come on during the same and disturb them.

Phenomena indicating an involvement of the stapedius muscle are rare,—*e.g.*, noises in the ear, which may occasionally be heard by others. The velum is rarely involved in pure cases of facial spasm ; in the few cases in which twitchings of the uvula were observed, as in a case described by Leube and Schütz, it was probably a complication.

In the cases of velum spasm (spasmus palatinus) which I have observed, an organic cerebral disease was almost always present,—once a cerebellar tumor, and once it was a residual symptom of an epidemic cerebrospinal meningitis. The vocal cords, or the internal laryngeal muscles, twitched synchronously with the velum. Twitchings confined to the velum, combined with crackling noises, have also been observed (Rosenthal, Williams, and others). Their description does not belong here. Siemerling and I have observed isolated twitchings of the velum in aneurisms of the vertebral.

Vasomotor, trophic, and secretory disturbances are usually absent.

We often find signs of another nervous affection accompanying a facial spasm, particularly hysteria, neurasthenia, epilepsy, hemicrania, and the psychoses. An hysteric type of facial spasm may also occur under the type of a glossolabial hemispasm. Blepharospasm and blepharoclonus are also often hysteric in character. I have observed a convulsive tic in the course of multiple sclerosis, probably due to the sclerotic process.

The course is generally a chronic one. It may continue during the lifetime of the patient. It runs, as a rule, an *intermittent* course, periods of rest and apparent recovery occurring. Strümpell observed remissions during pregnancy.

The prognosis is not favorable. Some cases, it is true, recover spontaneously or are cured by the treatment, but this is rare. Remissions are more often observed. If the disease has once become well rooted, the chances for its recession are very slight. That even in such cases we cannot speak of incurability is shown by the following case of mine : a woman who had a facial spasm of a persistent type for twelve years, which did not respond to any treatment, recovered completely from the effects of a sudden and continued favorable change of circumstances in her family. Blepharospasm has a relatively good prognosis.

Treatment.—The cause should always be sought after, special endeavors being made to ferret out those factors which produce it from reflex action. These include lesions in the trigeminal area, diseases of the ocular and nasal mucous membranes, carious teeth, diseases of the maxillary bones, etc. I saw a spasm which involved the facial and, to a lesser degree, the masseters disappear immediately after the extraction of a carious tooth. In one case the facial spasm, which had existed for a year, was said to have been eradicated by removal of a growth of the inferior turbinated bone. Although we have but little reliable data concerning the connection between diseases of the internal organs and facial spasm, it is desirable to investigate such disorders also, particularly bringing the genital organs into the field of examination. Hirt, for instance, saw a blepharospasm recede after treatment of a uterine flexion.

Generally, however, all of these attempts are futile. It is then advisable to pay attention to the general condition and to combat the neuropathic disposition by appropriate measures (hydrotherapy, climatic and diet "cures," etc.). Psychic outbursts should be avoided as much as possible by the patient, perhaps by changing his environment.

Direct Treatment.—Drugs do but little good, although the administration of arsenic and the bromides may be beneficial. Quinine, atropine, cannabis indica, tincture of gelsemium, etc., are almost always inefficacious. The use of morphine is rarely advisable.

Diaphoretic measures are suitable in recent cases; revulsives in the form of a cantharides plaster behind the ear may be used. *Electrotherapy* is of decided benefit, especially the galvanic current. Many methods of treatment are recommendable: anode of about ten centimetres square upon the nerve trunk, cathode upon the back of the neck or any indifferent place, weak currents of from two to three amperes, slowly turning on and off; anode upon the occiput, cathode upon a distant point; both electrodes upon the mastoid processes; anode upon the different branches of the pes anserini major, etc. If pressure-points are present, it is advisable to apply the anode to them in order to diminish their excitability. Even the application of the anode to the opposite parietal bone—*i.e.*, over the cortical centre for the facial—has been recommended. The use of a rapid vibratory faradic current has also been advised; likewise the static breeze.

In stubborn cases operative measures have been undertaken. When the *supraorbital* was sensitive to touch, and pressure upon this nerve inhibited the spasm, it was resected. A lasting good result was obtained in several cases. Simply cutting the nerve does not seem to be of any benefit. In other cases, the facial nerve itself was stretched. The result was a paralysis in place of the spasm, and with its disappearance, generally after several months, the spasm again came on (Bernhardt). In only one case was a recovery noticed (still present two years after the operation). In particularly stubborn cases in which it is a boon to the patient to be free from the bothersome twitchings for even a few months, this procedure may be tried, being careful to call attention to the fact that a paralysis will replace the spasm. In such cases a revulsive treatment (seton in the nuchal region, application of a cautery along the vertebral column) may be efficacious. That even in severe cases the possibility of a spontaneous recovery should not be ignored, is seen from the case I have described above.

Blepharospasm requires the treatment of the ocular trouble. *Cocaine* instillations may do good in such cases. The sudden immersion of the face in cold water may temporarily relieve the spasm. As this spasm is often hysteric in nature, every sort of suggestive influence may prove serviceable, as in a case of mine in which the trouble had lasted for twenty years. In addition, what has been said in general concerning facial spasm is applicable in such cases.

MASTICATORY SPASM.

Spasms confined to the motor trigeminal nerve are not rare. Both a clonic and a tonic form occur. In the former type, the jaw is tightly closed, the masseters and temporal muscles are prominent and feel as hard as a board. The patient cannot or can only slightly separate his teeth; passive attempts to separate the jaws are strongly resisted by the muscles. If the spasm lasts very long, the general health of the patient suffers from insufficient nourishment. A lateral displacement of the lower jaw occurs but rarely, and in unilateral spasms of the pterygoids the lower jaw deviates to the opposite side. *Trismus* is a symptom of *tetanus*, *meningitis*, is more rare in *tetany*, and occurs for a time in the tonic stadium of an epileptic attack; in diseases of the pons, at the onset of acute bulbar paralysis and also in tumors, a tonic masticatory spasm has often been observed. There is no doubt that it may be evoked by stimulation of the cortical centres (Lépine). It is rarely an *isolated* symptom. It is generally of *reflex* origin, due to inflammatory conditions of the temporomaxillary articulation or of its vicinity or of the mucous membranes of the inner cheek, to a periostitis of the jaw, to a carious wisdom tooth or to difficulty in its breaking through. In one case which I saw, a wound infection had preceded it; the trismus seemed to be the only symptom of a tetanus.

Trismus may also come on in the course of *hysteria*. Unilateral spasm of the pterygoids was observed by Leube in a girl suffering from hysteria and chorea. In nervous individuals it may be produced by a violent emotional outburst (terror). Some observations tend to show a rheumatic origin of these spasms. Kocher desires to separate trismus from hysteric spasms, calling the first an idiopathic spastic neurosis.

The clonic spasm produces rhythmical movements of the inferior maxilla in a vertical, and only rarely in a horizontal, direction. They may at times be so severe as to produce chattering of the teeth similar to that observed in ague. The masticatory muscles are also generally involved in general convulsions (hysteria, epilepsy, etc.); the tremor of paralysis agitans also occasionally localizes itself in this region.

A clonus limited to the masticatory muscles is a rare phenomenon. It has repeatedly been observed in hysteric individuals, generally as a temporary symptom.

I treated a young girl who for many years was troubled with persistent clonic twitchings of the maxillary muscles. It increased in speech so much that she spoke like one who had the ague; nutrition was also impaired.

A mild form of this spasm, gnashing of the teeth, often occurs in nervous children, and also in adults during sleep. Spasms of the muscles, drawing down the inferior maxilla, have been occasionally observed. I saw it follow a paroxysm of trigeminal neuralgia.

Spasms are also said to occur in the tensor tympani muscle and cause an aural noise; the movement of the tympanum may be detected by an otoscopic examination.

The prognosis is good, provided an organic disease can be excluded. It generally disappears after some weeks or months, though obstinate cases occur.

The treatment depends upon the cause. All inflammatory processes and other irritative conditions of the trigeminal area should be combated. If the irritation proceeds from a diseased tooth or from an ulceration within the oral cavity, the tooth should be removed or the latter treated appropriately. If the trismus renders access to the mouth difficult, narcosis is in place. A trismus of long duration demands artificial nourishment.

In recent cases, in which rheumatic influences may be at work, diaphoretics and counter-irritants to the mastoid process, or a cautery to the back of the neck, are advisable.

In addition, the different sedatives and nervines may be prescribed. Galvanic currents have also been found to exercise a curative influence.

I saw one case of hysteric trismus relieved by ovarian pressure.

SPASMS OF THE HYPOGLOSSAL REGION; SPASMS OF THE TONGUE; GLOSSAL SPASMS.

The general spasms of epilepsy and hysteria may involve the glossal muscles. They are also affected in chorea, while an isolated spasm of the muscles of the tongue is rarely seen. They may be *tonic* or *clonic*; a mixed form has also been observed.

In the tonic type, the tongue becomes hard and firm, its diameter decreases, and it is pressed against the palate or teeth. Speech and deglutition are hampered during the spasm, the respiration rarely so. The hinderance to speech, which is due to a tonic contraction of the muscles of the tongue of sudden onset and rapid disappearance, is called *aphthongia* (Fleury). The observation of Lange, that the tonic glossal spasms disappear during speaking and eating, is uncommon. The clonic spasms draw the tongue in and out of the mouth rapidly or slowly, or may move it from side to side around its longitudinal axis within the mouth. Generally both halves of the tongue are equally involved, though a hemispasm may occur, for instance, in hysteria, causing it to deviate to one side. A combination of unilateral glossal spasm and fibrillary tremor of the muscles has also been observed. The spasm may also extend to neighboring muscle regions, particularly to the lower facial.

The spasms are only rarely continuous. The attacks generally come on every few weeks, or they may occur as often as twenty or thirty times a day, rarely in the night, or every five to ten minutes. Each attack may last only a few seconds or minutes or persist for hours. It does not always cease during sleep. Glossal spasm is occasionally produced by peripheral irritation, inflammatory processes in the mouth, carious teeth (Mitchell), extraction of teeth, neuralgia lingualis, etc. As a rule, however, such causes cannot be detected. A neuropathic diathesis plays a prominent part in the etiology. It occurs often after *hysteria* and *hypochondriasis*, and in individuals who have formerly suffered from epilepsy or are epileptic.

The neuropathic diathesis shows itself also in combinations of glossal spasms and *psychic anomalies*, as I have observed several times. In a hypochondriacal, weak-minded individual, the spasms occurred in the muscles of the tongue, jaw, and lips at the same time; in another case, the tongue and masticatory muscles were alternately affected, etc. Sepilli observed it during a puerperal psychosis. Strümpell describes an occupation spasm of the tongue in a horn-player. Paresthesias and pains in the tongue may precede the spasm.

The spasm may be incited by emotion. In one case every attempt at mastication started it.

The prognosis is not unfavorable. The spasm may last for months or years, but in a majority of the cases recovery finally results.

Treatment.—The constitution should be strengthened by careful attention to diet, exercise, and hygiene. Sedatives should be employed in addition. All possible reflex causes must be treated. Galvanism may do good, using it in the manner described in the previous chapter (anode to the hypoglossal nerve). Operative procedures are hardly necessary. In Lange's case the hypoglossal nerve was stretched and then resected, and afterwards the genioglossal muscles were divided. The latter operation succeeded.

SPASMS IN THE REGION INNERVATED BY THE GLOSSOPHARYNGEAL NERVE; DEGLUTITORY SPASM; PHARYNGISMUS.

The spasms rarely reach the dignity of being styled a separate disease, but generally occur in hysteria, or as a symptom of hydrophobia and tetanus. They may also occur as pharyngeal crises in tabes. I observed a similar phenomenon in a case of bulbar gliosis. Local diseases of the pharynx or esophagus may produce similar spastic conditions.

SPASMS OF THE MUSCLES OF THE NECK.

On account of their persistency and injurious influence, these spasms constitute one of the most severe forms of spasm.

They may be unilateral or bilateral, confined to a few muscles of the neck, or involve many muscles of one side or of both sides. Those innervated by the accessorius nerve are particularly often affected, though it is not necessary to discuss these separately as "accessorius spasms," as they do not differ in symptomatology, prognosis, or treatment. The *sternocleidomastoid* may, for instance, be attacked alone or in conjunction with the *trapezius;* the spasm may involve the sternocleidomastoid and *splenius* of the same or opposite side, the splenius and trapezius, extend to the *scaleni* and *deep cervical muscles,*—in short, every possible combination may occur. The platysma myoides and omohyoid are also occasionally affected. That these spasms do not tend to limit themselves to a definite area is also seen by the tendency they display to begin in one muscle and extend to others during the course of the disease. *Neuropathically* and *psychopathically* tainted individuals are especially prone to be attacked.[1] The varied combinations of these spasms with other *neuroses,* and *espe-*

---

[1] In one of my cases the grandparents were related, the grandfather was a diabetic, the grandmother mentally diseased, the mother nervous, and the other children epileptic or insane. The patient had a general tic during childhood, and after marriage an acessorius spasm came on.

cially the psychoses, also indicate this. The so-called stigmata of degeneration are particularly frequent in these individuals.

Rheumatism of the muscles of the neck, *rheumatic torticollis,—i.e.*, a wry-neck produced by a rheumatic or myositic affection of the sternocleidomastoid,—also produces a stiff neck, but is not similar to these spasms in the narrow sense of the word. Congenital wry-neck will not be discussed here.

In only a few cases could a *trauma* be adduced as a cause. *Reflex* causes are also rarely found, for the muscular contraction occurring in diseases of the cervical vertebræ (especially caries) is an *accidental* condition, and has nothing in common with the cervical muscular spasms *sensu strictiori*. The same is true of those rare cases in which an ocular muscular paralysis causes a secondary rotation of the head and then a chronic torticollis (Nieden).

The torticollis occurring in ear disease, generally due to the extension of an inflammatory or suppurative process, rarely to reflex muscular contractions, is not an independent disease.

Organic cerebral diseases may produce spasms of the cervical muscles, though this symptom occurs rarely in them; I observed it once in a case of cerebellar tumor which pressed upon the medulla oblongata and nerves arising there, and several times with cysticercus cerebri.

It appears that *intoxications* may also play a part in the etiology. I have observed it result from alcoholism and chronic metal intoxication. In one intermittent case *malaria* was the cause, and the administration of quinine brought recovery. In one of my cases, the spasm followed *influenza*. It has also been observed after typhoid and pneumonia. Exposure to cold, traumata, and over-exertion have been frequently given as causes.

Some cases do not reveal any cause. "Bad habits" have been blamed, though it seems to me that such habits, or at least the transition to a chronic disease, could only occur in neuropathic individuals.

Symptoms.—We find a *tonic*, a *clonic*, and a *mixed* type of these spasms. Clonic spasms are the most frequently noticed, with a tonic muscular contraction occurring occasionally. A persistent tonic spasm of the sternocleidomastoid, causing contracture and wry-neck, is rarely a primary spasm, but is more often due to rheumatism, or is a result of disease of the cervical vertebræ and the surrounding parts, or to a traumatic myositis with fibrous atrophy, or is due to a *congenital* shortening of the muscle.

The symptoms are dependent upon the *localization*, the form, and the *intensity* of the spasms. If only one sternocleidomastoid is affected, the face is turned towards the other side, the ear approaching the clavicle, and the chin elevated; in spasm of the left sternocleidomastoid, the face is directed upward and to the right, while the left ear approaches the inner extremity of the left clavicle. If the twitchings are weak, only slight rotatory movements are noticeable. In addition to the sternocleidomastoid, the trapezius of the same side is often affected, almost always only the *upper portion;* the head is then likewise rotated to the other side and at the same time drawn backward, so that the occiput approaches the scapula. The contractions may occur simultaneously or alternately in the two muscles. If both trapezius muscles are involved, the head is simply drawn or thrown backward.

A very uncommon form and extension of the spasm was observed by me in an otherwise healthy individual, who had had the same trouble some years previous. The middle fasciculi of the trapezius were particularly involved by a contraction which attacked each fasciculus of the muscle, one after another, simulating a kind of muscular division (*myoschisis*). (Fig. 278.) The spasm differed from a fibrillary tremor in that the fasciculi, not the fibrillæ, were affected; and these not by a continuous wave but by a short contraction, a tic. Another peculiarity was the fact that every time the shoulder was elevated or extended forward this region, and particularly that of the scapula, was the seat of a marked temporary rapid contraction, sometimes occurring on both sides.

The spasm confines itself rather often to the *splenius;* the head is drawn backward and at the same time rotated to the side on which the spasm is located.

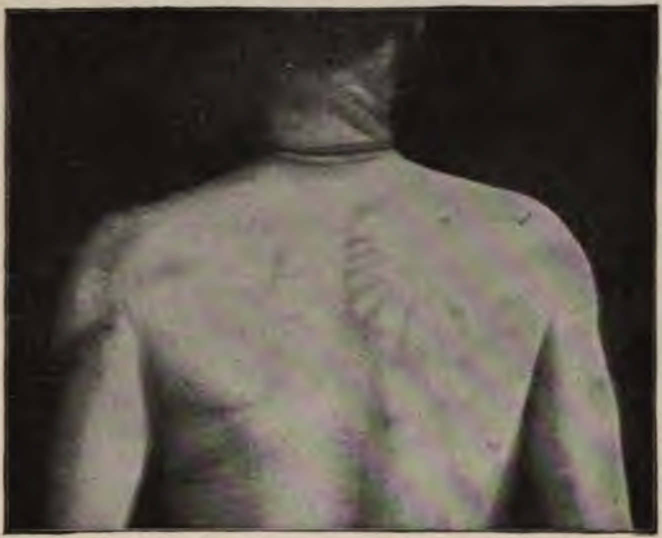
The spasm sometimes consists in simple nodding movements,—*nodding* or *salaam spasms* (*spasmus nutans*),—occurring especially in small children at the time of dentition. These are due to clonic contractions of the deep cervical muscles (*recti capitis, longus colli,* etc.); the sternocleidomastoid may also be involved.

These nodding spasms, first described by Barton, Bennet, and Newnham, are generally combined with nystagmus, occurring especially when the head of the child is fixed. *Strabismus* and *blepharospasm* may also be present. The disease, which may also be a family one, attacks children in the early years of life. The simple *rotatory spasms* or tics, which occasionally occur, are due to a spasm of the *inferior oblique.* The involvement of the vocal cords and palate in spasms of the accessorius, as observed by Gerhardt, is unusual.

The intensity of the spasm varies. It may be slight or severe. The supraclavicular arteries and nerves may be compressed in the latter case (Romberg). An *hypertrophy* of the muscles attacked sometimes occurs. It has been claimed that an atrophy of the muscles of the opposite side may be the cause of the spasm (Féré).

The contractions are *increased* by *emotional* excitement, introspection, and by efforts to overcome the spasm; and are decreased by physical and mental rest as well as by distracting the patient's attention. Brissaud, who lays stress upon the psychogenic origin of the disease,—he speaks of *mental torticollis*,—calls attention to the fact that some patients may stop the spasm by merely laying the finger on the chin.

FIG. 278.



Spasm of the right trapezius, especially of its middle portion.

The spasm in some cases is chronic, although remissions and exacerbations occur; in others it occurs in attacks, between which complete rest reigns. Ten to thirty contractions may take place in a minute. *Sleep* does not always cause a cessation of the spasm.

Grassett has lately described a complicated spasm under the name of "*tic de colporteur*," or "spasme polygonal," in which the head is inclined towards the left shoulder and turned towards the left, the left arm resting on the body, the right drawn backward. He refers the tic to the occupation of porter, from the automatic movement in carrying a burden upon the left shoulder. The designation "spasme polygonal" is founded upon such an artificial basis that it need not be discussed.

Pain is generally not present, but a drawing pain in the nucha may precede the contractions or accompany them. Paralytic phenomena are not present, nor are any defects of sensation or of the special senses.

That *psychic disorders* are often combined with these spasms has been commented upon before. Romberg cited a case of Brodie in which a psychic disturbance to a certain extent formed the equivalent of the spasm of the accessorius, and which ceased during its occurrence. I observed a similar *alternation of spasm of the cervical muscles* and *hallucinatory confusion.* In another of my cases the spasms developed after delirium (probably of alcoholic origin). In a case of Gowers's *melancholia* existed ten years before the onset of the spasms.

Differential Diagnosis. — *Rheumatic torticollis* differs from true spasm of the cervical muscles in the severe pain and sensitiveness of the muscles on pressure and passive movement. Torticollis existing from birth may be distinguished by its congenital development, secondary alterations of the cervical vertebræ, shortening of the muscles, and mechanical fixation of the head.

In chronic wry-neck a *local disease,* especially disease of the cervical vertebræ, should be thought of. It should also not be forgotten that organic cerebral diseases (especially a tumor or cysticercus) may be the cause. A close examination and search must be made to exclude these conditions.

Myoclonia and *maladie des tics convulsifs* (general tic) may commence with spasms of the cervical muscles. In the latter, however, a tic of the facial muscles, especially the orbicularis palpebrarum, occurs. It must be remembered that a *facial spasm* may be combined with a spasm of the cervical muscles without a condition of general tic developing from this condition. As soon as sytematic movements or echolalia, etc., occur, a diagnosis of general tic can be made. Myoclonia, it is true, involves also the cervical muscles; it is, however, not confined to them, and does not, as a rule, restrain their movements.

Ordinary *chorea* can easily be differentiated from this condition, though they may be combined or follow each other.

Pathology. — No alterations of the central or peripheral nervous system have ever been observed. The disease is due to *irritation of the nerve nuclei* or *nerve centres.* It is probable that the cortex and the kinesthetic centres for the cervical muscles are the starting-places for the spasms in most cases, and that a generally inherited instability promotes their onset.

Prognosis. — It is generally grave. Spontaneous recovery only occurs in mild cases. Under treatment I have observed not a few of these spasms disappear. The disease, however, generally lasts years or a whole lifetime; it attains a certain degree of severity and then remains stationary or varies in intensity. Those spasms occurring in young children often get well; it is perhaps the rule for typical cases to recover, especially with the cutting of the teeth (Henoch, Dickson). On the other hand, in a number of cases a relationship between these spasms and epilepsy, idiocy, and severe lethal cerebral diseases has been observed (Hochhalt, Böhr), though it is doubtful whether in these cases it was an ordinary spasmus nutans. In those cases where hysteric phenomena appear in addition to the spasms the outlook for complete recovery is good. I have lately seen some severe cases of accessorius spasm recover after persisting for years, and under such different forms of therapy that I could not help thinking of a psychic factor. The condition may become so grave as to lead to attempts at suicide.

Treatment.—If the cause can be found, treatment should be directed against it. In this manner dietetic procedures, iron, hydrotherapeutic measures, etc., may be of advantage. The patient should especially be guarded from excitement, should avoid seeing strangers as much as possible, and should select a quiet place to live. If alcoholism or some other intoxication is the cause, the patient should be removed from the influence of these poisons. An occupation which does not demand too much exertion is permissible.

The *bromides*—*morphine* and *opium* more so—relieve the spasms, though these drugs, especially subcutaneous injections, should only be given as a last resort. Curare is said to have been successfully used in some cases. Tincture of gelsemium, valerianate of zinc, and extract of conium are praised by some. Romberg recommends oxide of zinc for the nodding spasms. *Supporting apparatus* may do good, the purpose of which is to press against the head in a direction opposite to that of the spasm. Tight bandages should never be used.

Electricity has produced good results in some cases. If pressure-points can be found,—they are rare in this disease,—the *anode* should be applied to them ; otherwise the *accessorius* nerve should be brought under the influence of the anode, while the cathode is placed upon the muscle. *Faradization* is also of benefit. French authors recommend faradization of the sternocleidomastoid of the opposite side, which is generally atrophic. Vigoroux and Charcot have reported good results from it.

Definite observations do not exist as to the benefits of *massage* and *gymnastics*. *Counter-irritants* have done good in not a few cases, fly-blisters, the seton, and the cautery having been used.

The results of operative procedure are doubtful. Section of the tendons of the muscles attacked by the spasm does not do any good. *Section*, *stretching*, and even *resection* of the accessorius (neurectomy) produce only rarely any lasting good results. The latter operation is the best of the three. Petit and others reported recoveries after these operations, though the time of observation was too short. Francis observed after section of the accessorius, first, recovery, then a relapse with mental disturbances, and, finally, recovery.

I observed in many cases that the spasm, when prevented from reaching the originally attacked muscles, would spring over to others: the *central irritation* remains and is discharged by other paths. In several cases the upper cervical nerves were divided, in addition to the accessorius, and with good results. In other cases the posterior roots of the upper cervical nerves were cut, with a favorable termination. In one of my patients the tendons were cut, then the accessorius was stretched, and later, section and resection of it were undertaken. Notwithstanding a complete atrophy of the sternocleidomastoid and a partial atrophy of the trapezius, the spasms increased in intensity in the splenius, the omohyoid, and the remainder of the trapezius. I next applied a seton, also without any result. A cold-water treatment following this gave the patient the best results.

All the above operations seem to me to act merely by *counter-irritation*. One fact, however, is opposed to this idea,—*i.e.*, the results of the twelve cases operated upon by Kocher and Quervain, in which the sternocleidomastoid, trapezius, splenius, complexus, and inferior oblique were divided. Seven were cured, though the recovery did not seem to be a complete one in every case. Brissaud, on account of the psychogenic origin which he claims for it, argues against operative methods.

Collier lays bare the accessorius and compresses it with a silver ligature.

Corning recommends a peculiar method, which he calls *elæomyenschisis*. He injects into the affected muscle an oil which becomes solid upon cooling (ol. theobromæ and paraffin) and with an ether-spray freezes it (!).

In one case which I saw, warm sea-baths were said to have produced a cure.

All in all, we are still groping in the dark in our treatment of these conditions.

SPASMS OF THE MUSCLES OF THE TRUNK AND OF THE EXTREMITIES.

If we exclude the spasms of the trunk and extremities occurring in the different organic diseases of the brain and spinal cord, and the motor neuroses in which spasms occur more or less in the entire muscular system, or in those muscles which are used in certain occupations, *idiopathic* and *localized* spasms of the muscles of the trunk and extremities are rare occurrences. They have, however, though only in isolated cases, been observed in all the muscles of the body. They may attack single muscles as the levator anguli scapulæ, the latissimus dorsi, the deltoid, or the same muscle on both sides, as the pectorales, etc., or a number of muscles in various groupings, as the levator anguli scapulæ with the rhomboid, the triceps with the pectoralis major, etc. Occasionally the spasm occurs in all the muscles innervated by the same nerve or pair of roots. I observed in one case clonic twitchings in the deltoid, biceps, brachialis internus, and supinator longus, which succeeded one another in the manner noticed in electric stimulation of Erb's point. In a case of Laquer, the clonic twitchings were confined to the muscles innervated by the musculospiral nerve; in another case (Hochhaus), only the triceps and supinator longus were affected. Schultze has described a triceps spasm.

Tonic spasms have been repeatedly observed in the shoulder-muscles (rhomboid, levator anguli scapulæ), in the flexors of the lower arm, hand, and fingers, and also in the interossei. Contracture may result from it. W. Mitchell described a case in which the spasms were so severe that the nails bored into the palm of the hand. The *calf-muscles* are the most frequently attacked of the muscles of the lower extremity, though the tonic contractions may occur in all the other muscles. There developed, for instance, in a neuropathic patient of mine tonic spasms in the right psoas and sartorius, which lasted half an hour and occasionally extended to the rectus femoris and gracilis. A physician who had this trouble came to me with the fear that he had Thomsen's disease.

In one case in which the tensores fasciæ latæ, the extensors of the thigh, and the recti abdominis were affected for six years, I saw a pronounced hypertrophy ensue. Tonic cremasteric spasms were described by Berger.

Tonic and clonic spasms have also repeatedly been observed in the quadriceps; in one case they occurred only in walking. The adductor femoris and the rotators seem to be less often involved. Spasms of the flexors of the lower leg seem also to be rare.

Rhythmical clonic twitchings confined themselves in one case to the iliopsoas, and later, the same muscle of the opposite side. In another case (Jobert) the peroneus brevis, and in a third (Bernhardt) the peroneus longus and brevis were affected. In the last case they persisted during sleep. Movements of the tendon in the groove behind the external malleolus produced a crackling sound. Manipulations which inhibit the spasms (compression and extension of the tendons) tend to cause the spasms to spring over to other muscles. Clonic spasms occasionally occur in the quadriceps and also in the tensor fasciæ latæ. The muscular movement (myokymia) described by Kny and Schultze—a persistent and pronounced fibrillary tremor and swell of the muscle, which may be painful and combined with hyperidrosis—may, perhaps, be included here.

Most of those affected are very nervous. Often *hysteria* is present, or some similar condition. In others the diagnosis of *neurasthenia* could be made, or a marked neuropathic predisposition was evident. In one case, *epilepsy* had preceded the spasm; in another, *writer's cramp;* in another, *chorea;* etc. The tonic spasms, however, are more often due to *over-exertion*, *local irritation*, varicose veins, and intoxications (diabetes, alcoholism, cholera, etc.).

Trauma is an important etiologic factor in these localized tonic and clonic spasms. They are a direct result of the trauma, or are due to an irritation arising from the wound or cicatrix. The trauma has this effect in congenitally nervous individuals. In Laquer's case, massage used to relieve a writer's cramp acted as a trauma. Local muscular spasms have also been repeatedly observed in cases of articular inflammation, and their reflex origin in such cases could not be doubted. In accord with this, is the fact that neuralgias as well as pains arising in the amputation-stumps may evoke local spasms. Though the central nervous system, so long as it is healthy, does not react in this manner to pain, the continued presence of these spasms may entail a central diathesis.

The prognosis as to recovery is a doubtful one. Ordinary spasms in the calf-muscles generally disappear of themselves, but may be very stubborn. All these spasms may last months or years; but, as a rule, they disappear sooner or later and often suddenly.

Treatment.—The methods which have cured patients are various in kind,—electricity used in many ways, cold douches, tenotomy, threats of an operation, in short, the most *heterogenous methods of treatment*. This shows the *psychogenic* character of the spasms and the curative effects of suggestion. It does not, however, always cure, and it is not proven that all remedies act in this manner. *Local massage*, *wet packs*, *warm baths*, *stimulating ointments*, *sinapisms*, and electricity, are all of benefit. It is important that the patient should avoid all over-exertion. Spasms which occur in a definite region often subside after a galvanic anodal treatment. It is apparent that the general nervousness which is present demands a constitutional treatment.

RESPIRATORY SPASMS.—*Tonic spasm of the diaphragm* occurs rarely. In it the epigastric region is raised, the lungs extend lower than normally; abdominal breathing is absent, while the upper thoracic regions are set in motion by the rapid dyspneic breathing. The patient feels a want of air, and generally experiences some pain at the insertions of the diaphragm. The diaphragmatic movements are absent during respiration; the so-called *acute lung inflation* may be a result of these cramps. It may be due to *hysteria* and disappear quickly and spontaneously, or in milder cases persist for some time. In other cases it becomes an earnest, anxiety-producing disease, evoking severe asphyxia, and should be combated with *cold douches*, *the patient seated in a warm bath*, *hot cloths upon the epigastrium*, *faradization*, *stabile anodal treatment of the phrenic*, and, as a last resort, *morphine* and even *chloroform*. The spasms of tetanus and tetany may also attack the diaphragm. *Clonic diaphragmatic spasms* (*singultus*, *hiccough*) occur much oftener and are known to all from personal experience. Brisk contractions of the diaphragm produce an *inspiratory noise*, the glottis not dilating at the same time. It seems to me that the muscles of deglutition are contracted simultaneously. In mild cases, in which the spasms rarely follow, and if they do are not severe, the individual (we cannot say patient) is not much troubled. In severe cases, in which other respiratory muscles are also involved, and in which a hundred or more spasms may occur in a minute, the trouble is very annoying, and hinders speaking, eating, and breathing.

The chief danger is in its persisting and lasting months or longer.

Of such a stubborn character is often the *singultus* in hysteric patients. It occasionally constitutes an ominous sign of an *organic cerebral disease* (apoplexy, tubercular meningitis), and occurs especially in the agonizing stage.

It is also said to occur from *direct stimulation of the phrenic*. There is no doubt that the spasm is sometimes produced by irritation arising in the *sexual* sphere or in the *gastro-intestinal* tract. Excitement may also cause it; psychic infection is often a factor (Bernhardt).

Other types, as *yawning spasms* (oscedo, chasma), *sneezing spasms* (ptarmus, sternutatio convulsiva), can hardly be called diseases. They occur episodically in the course of hysteria and in nervous individuals, and never reach a tormenting intensity. Yawning spasms may also be an aura of an epileptic attack. Spasms of yawning are also occasionally seen in organic cerebral diseases, especially tumors and abscesses of the cerebellum. Recently a woman, who had a general neurofibroma, was sent to me on account of a *snoring spasm* (rhenchospasm); she constantly, and against her will, emitted sounds similar to that observed in many individuals during a deep sleep. Its psychic origin was evident in that, by distracting her attention, the snoring ceased. Treatment did some good, although she is still under my charge.

Singultus is occasionally prevented by holding the breath, by fright, by distracting the attention, etc. If stubborn, *counter-irritants* and sedatives are in place. *Bismuth subnitrate* was successful in one of my cases. Faradization and galvanization of the phrenic nerve, passing a sound down the esophagus, compression of the lower part of the thorax, and many other methods have been recommended.

The spasms which involve all or almost all the respiratory muscles and which cause forced respiratory movements in rapid succession are usually *hysteric* in their nature (see page 673).

A very peculiar case, which I also saw, has been described by Edel: a man who for years had had respiratory spasms, which occurred upon every touch, noise, etc., accompanied by pronounced dyspnea (contraction of the auxiliary muscles). Except an increase of reflex excitability nothing was found. Schapiro described a unilateral clonic spasm of the respiratory muscles. Purely expiratory spasms, due to a spasmodic action of the abdominal muscles, may also occur.

Spasms of coughing are generally hysteric. They may be reflexly produced from the stomach, external ear, nose, and perhaps from the liver and spleen.

SALTATORY REFLEX SPASM (STATIC REFLEX SPASM).—This peculiar type of spasm was first described by Bamberger. As soon as the patient touches the floor with his feet he indulges in hopping, running, and dancing around, produced by clonic contractions of the muscles of the lower limbs, especially in the calf-muscles.

These spasms disappear when a recumbent position is assumed. Other phenomena are absent, or we find signs of general nervousness and of hysteria combined with the spasms. The skin and tendon reflexes are generally increased, and some (Erlenmeyer, Kast) believe that this increase of the deep reflexes has much to do with the spasms. It occurs in both sexes and at every age, without cause or after some emotion or an *infectious disease.* It is probably not an independent form of spasm, but only a symptom or a rare form of *hysteria.* It also may develop in ballet dancers as an occupation neurosis.

The prognosis is good. It generally recedes after some months. The use of sedatives, electricity, wet packs, anesthetizing the soles of the feet, etc., are advisable. Gowers recommends a diaphoretic treatment.

MALADIE DES TICS CONVULSIFS; GENERAL TIC; MALADIE DES TICS IMPULSIFS; MYOSPASIA IMPULSIVA.

This rare motor neurosis, which has particularly been described by Guinon and Gilles de la Tourette, is characterized by the following phenomena: (1) twitchings of the facial muscles, (2) systematic movements, repeating themselves in the same manner, (3) echolalia and coprolalia, also echokinesis, and occasionally (4) imperative or fixed ideas and movements.

This disease generally occurs in *children* between seven and fifteen years of age and who are neuropathic by *heredity.* As a rule, it is a general or polymorphous neuropathic diathesis; only in one case could I discover a direct heredity, in which the grandmother had had the same disease, and three of her daughters and three grandchildren had inherited it. A brother was an epileptic.

A sudden emotion, a trauma, or an infectious disease is the most common exciting cause of the first attack.

The first symptom noticed is generally *twitching of the facial muscles,* particularly a blinking of the eyes, drawing up of their corners, distortion of the mouth, or a rapid opening and closing of it, etc. Twitchings of the *muscles of the neck* come on afterwards. In its further course, often only after years, movements occur which give the impression that they are for a *definite purpose*, or to produce an *effect*, or are merely the result of *habit*. The patient grasps his nose, pulls his hair, strokes his chin, throws his head to one side, claps his hands, stamps his feet, catches imaginary insects, imitates spitting, dances, hops or runs around, and the like.

These movements are repeated in a stereotyped manner. The patient is forced anew to bring his muscles into the same action, and originally judicious and physiologic movements become pathologic. Though all muscular regions may be affected, the face, neck, and upper extremities are particularly liable to be affected. Synergetical muscular action is rare, as are also single movements. It is generally a co-ordinated tic. They differ from voluntary movements not only through their seeming absence of purpose and constant repetition, but also by the short, rapid, and forcible nature of the muscular action.

Articulation, phonation, and respiration are generally involved; the patient is forced to make inarticulate sounds or even words. It is often a simple smacking of the lips or a chuckling, more often *imitation of the voices of animals*, sometimes senseless words (one of my patients constantly repeated, "kritsch, kritschkratsch, quatsch, krum dum, krikideidei"), or more often ugly and obscene words (*coprolalia*), or references of a sexual nature. The impulse to repeat words or sounds (*echolalia*) or to imitate movements (*echokinesis*) is rarely present.

The patient, whose intelligence generally remains intact, suffers a great deal, later in life, from these mimic or habit spasms. Many by energetic will-power repress these twitchings for a time, but few are able to do so very long. A ballot dancer with tic was able to appear nightly before the public; a post-office official treated by me was able to carry on his duties without those around being aware of anything wrong; but this repression of impulses produces an inner unrest, and afterwards the movements break out with renewed vigor. This cessation of the tics during work is not merely due to self-control, but also to the *diversion of the attention*. If the patient is alone and unobserved, he may do some work without being disturbed by muscular spasms. As soon as he comes in contact with the outer world his motor apparatus starts in action, and the intensity of the twitchings represents the height of the excitement.

Voluntary movements generally still the tic, but if the patient is very excited, or if the imperative actions are of marked severity, they may be only momentarily inhibited.

The intensity of the tic varies; *remissions* may occur as well as a paroxysmal increase of the trouble.

In some cases *fixed ideas* rule the patient,—*e.g.*, necessity to count his footsteps or the windows of the houses, to taste everything, to walk backward, etc.

One of my patients felt impelled to slur the terminal r of words: he would say door-r-r-r. Another a mania for gathering together paper and food, which she had kept in a little sack; when filled, another was in readiness.

The patients know what they are doing, but cannot help it; if they combat it, they are restless till the impulse has been satisfied. The intelligence is generally intact, though it may be impaired later on from the persistent lack of attention. It is a *chronic* condition, lasting for years or the whole lifetime. Many authors, though wrongly, consider it incurable. I have seen typical cases recover completely. Charcot, as did I, observed long remissions. A majority of the cases are, however, chronic and progressive in character.

Developed cases are easily diagnosed. As long as the tic is confined to the facial muscles it may be confused with ordinary *convulsive tic*, though this is generally unilateral. If it extends to the muscles of the neck, the diagnosis is made probable, and it is confirmed when co-ordinated tics are observed.

The *systematic* character of the movements, the relatively *long pauses*, the stilling influence of work, the echolalia and coprolalia distinguish it from *chorea minor*. A combination of *maladie des tics convulsifs* with chorea also occurs. Spasms in *hysteria* occur suddenly, the stigmata of hysteria may be found, echolalia and coprolalia are generally absent, and the trouble may be stopped through suggestion.

Paramyoclonus differs materially from it. It is identical or similar to the conditions described as occurring in the jumpers (America), the Latah (Malay), and the Meriatschenje (Siberia).

Fixed ideas and actions occurring in paranoia are due to hallucinations, and thus differ from those observed in this disease.

Treatment.—Sedatives and nervines are generally useless. At the height of the paroxysms it may be necessary to administer chloral hydrate or chloroform to give the patient some rest. Arsenic has no influence. Hyoscine and eserine were also found by me to be of no benefit. *Isolation* of the patient is the best therapeutic measure. A mild hydrotherapy also does good.

Hypnosis is also useless; most of these cases cannot be hypnotized. In a few of my cases *gymnastics* produced good results.

PARAMYOCLONUS MULTIPLEX (FRIEDREICH); MYOCLONIA; POLYCLONIA.

This disease was first described by Friedreich in 1881. The observations made later by others are only partly similar to the original ones of Friedreich.

The disease is characterized by *clonic muscular contractions*, occurring mostly in the body and limbs, rarely in the facial muscles. The contractions are short, sharp, lightning-like, *unsystematized;* may occur symmetrically on both sides of the body; do not occur synchronously or rhythmically; may be disseminated or localized; and may involve a single muscle or a whole muscle-group. Although all muscles may be attacked, some are more so than others,—*e.g.*, the *supinator longus*, the *biceps*, the *trapezius*, the *quadriceps femoris*, the *semitendinosus*, etc. Active movements are not, as a rule, hindered, and have a restful, spasm-stilling influence; the opposite is true of the emotions. The twitchings are lessened if the attention is diverted. They disappear completely during sleep. The tendon phenomena are generally increased. Mere touch may increase the convulsions, the contractions in which may reach a rate of from sixty to one hundred a minute.

The motor phenomena of stimulation are generally the only symptoms noticed. All other functions are normal, even the mechanical and electrical excitability of the muscles. A combination of paramyoclonus and urticaria was observed several times by me.

In many cases a trauma, or a fright, or an infectious disease, preceded the trouble.

Unverricht has described a particular form of myoclonus, characterized by its combination with *epilepsy* and the *familial* nature of the disease. In some of his cases, the tongue and deglutitory muscles, as also the diaphragm, were attacked. Cases of this kind were also described by Weiss, Krewer, and Sepilli.

Males are more often attacked than females.

The difficulty of sharply separating and differentiating the neuroses from each other is particularly exemplified in attempts to establish the nosologic independence of paramyoclonus. As soon as something is taken away from Friedreich's original clinical picture, there is danger of entering the domain of another neurosis. Even the view that Friedreich's form is an independent disease has been doubted (Moebius, Strümpell), and some authors are inclined to regard myoclonus as merely a type of hysteria. I regard that as wrong, although an hysteric form of this disease exists. In some cases, as in those of Unverricht, the disease had a close connection with epilepsy; in others, all that can be detected is, that it resulted from psychic degeneration. The attempt to make this neurosis a modification of chronic chorea (Moebius, Böttiger) has not secured many adherents.

The non-hysteric cases of myoclonus form an independent disease; the cases of Unverricht, a particular type. It must be acknowledged that the hysteric character cannot always be easily recognized.

Henoch's chorea electrica is probably identical with myoclonus.

The prognosis in pure cases is a grave one. Friedreich believed he had seen cases recover; relapses, however, occurred, and the disease continued. In one case brilliant results were secured by electricity, but it was probably an hysteric case. Careful examination of all the nervous structures, which Schultze was able to undertake in one case, gave negative results.

Friedreich believed that the basis of this neurosis was a condition of excitation in the ganglia of the anterior horns of the spinal cord. Unverricht's views coincide. Wagner observed similar spasms in animals after extirpation of the thyroid gland.

Treatment.—Thyroidin is said to have cured one case; I found it of no benefit. Chloral hydrate and the bromides have a calming influence and produce a temporary betterment. Arsenic has done good in several cases. Galvanic currents may be tried.

THE OCCUPATION NEUROSES. (OCCUPATION SPASMS; FATIGUE NEUROSES; CO-ORDINATED OCCUPATION NEUROSES.)

We understand by an occupation neurosis a disturbance of innervation of the musculature, acquired by exercise, and occurring only in definite, complicated motions, while the muscles obey every other action of the will. The most common form, which we will take as a type of all, is

WRITERS' CRAMP (GRAPHOSPASM, MOGIGRAPHIA).—This denotes a disturbance of innervation of the muscles used in writing, in consequence of which writing is made difficult or impossible, and the handwriting distorted and indistinct, while the hand may be used for every other purpose. The exciting *cause* is over-exertion of the muscles by writing. The disease, therefore, occurs especially in those whose occupation demands a great deal of writing. They are, however, rarely individuals with an intact nervous system, but most of them are of a *neuropathic predisposition*. Almost all of my patients were *neurasthenic;* some were troubled with *hemicrania*, others with neuralgia; one had epilepsy, another stuttered, etc. Combinations of writers' cramp with *convulsive tic, agoraphobia, tabes*, etc., occur. The fact that *several members of a family* may be affected also serves to show its neuropathic origin.

Its onset is favored by the use of sharp, hard, steel pens and *improper* *methods of writing.* Especially bad is the method of using the little finger as a support to the hand and writing only with the muscles of the fingers. The more the small muscles of the hand are strained, the sooner does the spasm result. In a lady treated by me the trouble commenced after she had nursed her husband who had had paralysis agitans, and she had feared that she would become afflicted with the same disease. In another of my patients the writers' cramp occurred after a spiritualistic seance. I wish to state here that this incubator of psychic infection has furnished me with a number of cases of the psychoses and neuroses.

Male adults are most frequently affected, though even children are not immune.

Continued emotion may cause it. In a few cases it has occurred after injuries to the hand and after local inflammatory processes (Seeligmüller).

The disorder develops gradually. The patient first tires more easily in lengthy writing; he soon learns that he has not the same power over his pen as formerly, and that he cannot write as quickly or as smoothly. After some time he notices that the muscles contract spastically so that the pen-holder is held extraordinarily tight.

In most cases it is a spasm which produces the difficulty. The *spastic* form of writers' cramp is the ordinary form. The tonic spasm attacks particularly the flexors of the fingers, especially of the thumb and index-finger; the extensors are rarely involved. The fingers are sometimes forcibly separated, and the thumb drawn into the palm by the spasm. Gradually it extends to the muscles of the hand and forearm; the wrist-joint is over-extended, pronated, or supinated, and the hand raised from the paper. Writing also causes *pain* in the muscles, bones, or joints. The trouble gets worse until the patient is not able to write a single word, or until the writing becomes impeded and uncertain from the spasm. The writing appears irregular, zigzag, and coarse; some letters are too small, others too large, and some strokes are entirely lost. The more he observes his writing and the more it annoys him, the worse does it become.

We have also *tremulous*, *paralytic*, and *neuralgic* forms of writers' cramp, or combinations of two different types.

An objective examination reveals nothing. There is no disorder of motility or sensibility, no ataxia or tremor.

Pressure-points on the nerves are also rarely observed. Here and there we find a swelling and thickening of the tendon-sheaths of the extensors of the fingers (A. Pick), or periostitis of the external condyle of the humerus (Runge). Neurasthenic symptoms may be present,—increased tendon reflexes, vasomotor disturbances, etc.

The course of the disease is generally chronic. If the other hand be used to write with, it also will become affected after some time.

The prognosis is not very favorable. Only in a minority of cases did recovery take place; and in those apparently cured, *relapses* often occurred. The longer the affection has been present the less chance is there for recovery. The neuralgic form has a better prognosis.

Diagnosis.—Guard against diagnosing every disorder of writing as writers' cramp. Multiple sclerosis, tabes (when it commences in the upper extremities), a slowly developing left hemiplegia, paralysis agitans, etc., may produce disorders of motility, sensibility, and co-ordination, which at first are only recognized in the execution of complicated movements, as in writing. A careful physician will, however, easily detect that these symptoms appear also in the examination of motion, sensation, and co-ordination.

A certain difficulty is experienced in the diagnosis of cases in which a *hysteric* or *neurasthenic* tremor produces inability to write. That other phenomena of hysteria and neurasthenia are present does not speak against the occurrence of writers' cramp, as they are not rarely found together. If it is due to nervous tremor, the prognosis is much better than if it were an independent disorder. The nervous tremor is noticed in other movements, and the cramp in cases of this nature may be lessened by distracting the attention or by the influence of suggestion.

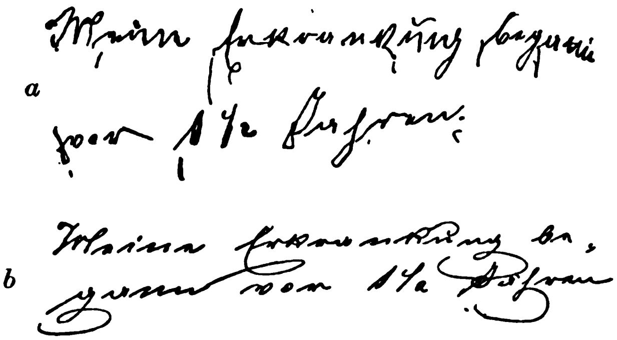
These signs are, however, not always present, nor are they always reliable. In severe, old cases of writers' cramp, the disorder is noticeable in other fine manipulations of the hand also; for example, in threading a needle, sewing, violin playing, etc.

Anatomic alterations have not been found and probably will not be in future cases. It is without doubt a *purely functional* disorder, an *exhaustion neurosis*, which has its seat in the centres for co-ordination, in the central apparatus which governs the associative acts of the muscles which are necessary in writing. The centrifugal and sensory excitation which accompany writing, and which are not perceived normally, now become apparent, and evoke a disinclination for writing, and the more the attention is directed towards these occurrences, the more severe does the spasm become.

Treatment.—The patient *must absolutely abstain from writing for a long time*. It should be ascertained whether the manner of writing is faulty, and if this is the case a *modification of the method of writing* should be recommended. If the children in school were from the first taught the proper way to write, the teacher would aid much in preventing writers' cramp.

Thick or cork pen-holders should be used, lead-pencils and a goosequills are preferable to steel pens, and the writing should be done in large letters in round script. Pen-holders with a sort of ring have been recommended. Nussbaum's bracelet aids a great deal with some. Zabludowski's pen-holders have also been praised. Each individual case must be studied by itself and mechanical appliances altered or improvised to suit it. If local alterations, as swelling of the tendon-sheaths, are present, the treatment must naturally be directed against them. Drugs are of little benefit, though sedatives and nervines, and lately an opium-bromide cure, have been recommended (Dornblüth).

FIG. 279.



Handwriting of a patient with writers' cramp, without and with the use of Nussbaum's bracelet. (After v. Limbeck.)

Hydropathic treatment may do much good, also a change of climate. This is particularly true of neurasthenic cases.

An electric treatment is of uncertain benefit. The galvanic current may be used, sent through the cervical cord or used by placing the cathode in the nuchal region and the anode in the neighborhood of the brachial plexus and the individual nerves of the arm. M. Meyer recommended a stabile anode treatment of possible pressure-points upon the vertebral column.

The best results have been obtained by *massage* and *gymnastic exercises*. In one of my cases the Swedish exercises were particularly of benefit. These consist in movements of resistance in the muscles of the hand and fingers. In many cases suggestion is a factor in the treatment. If all treatment is futile, a typewriter should be recommended for future work.

OTHER OCCUPATION NEUROSES.—These are almost as numerous and varied as are the kinds of occupations. We have piano-players', violinists', flutists', celloists', seamstresses', telegraphers', tailors', cobblers', cigar-rollers', watchmakers', milkers', blacksmiths', and dancers' cramps, and many others.

The facts given under writers' cramp for the course and symptomalogy apply here also.

Piano-players' cramp occurs especially in pianists during the hours of instruction. It consists, as a rule, in abnormal muscular contractions, from which a finger or several remain lifted from the keys or are pressed upon them, so that playing must be discontinued. The trouble is generally painful, and the pain is felt not only in the fingers and arms, but particularly in the region of the shoulders and the spinal column. A paralytic form, with complete paralysis of the hand or fingers, may also occur. In most cases we find only pain and exhaustion during the playing.

Violinists' cramp may affect the bowing hand or the hand pressing upon the strings, occasionally both. In one case which I treated, paresthesias came on at every attempt to play the violin, and rendered further playing impossible. As soon as the patient ceased playing all trouble disappeared. Another had pain in the muscles of the shoulder and upper arm.

Flute-players' cramp affects either some of the fingers or the whole hand, the left more often than the right.

Blacksmiths' cramp involves the muscles of the upper arm and shoulder, particularly the deltoid and biceps, which contract in the lifting of a hammer. The pain accompanying the cramp is very severe.

Milkers' cramp is a disorder which affects dairymaids who have milked cows day after day; every attempt causes a tonic spasm in the flexors and extensors of the hand and fingers accompanied by violent pain. The cramp is occasionally accompanied by a feeling of deadness and cold in the fingers. A combination of this spasm with a degenerative neuritis of the median has been described by Remak. Runge has described as waiters' paralysis a weakness of the extensors and supinators of the hand, which is produced in waiters by excessive carrying of plates, glasses, etc.

Seamstresses' or tailors' cramp affects especially the muscles of the thumb and index-finger; the cigarmakers' spasm, the hands during the rolling of cigars; while telegraphers' cramp becomes noticeable in jotting down the lines and dots of Morse's system.

I have seen in two cases a barbers' cramp (keirospasm or xyrospasm)—*i.e.*, a spastic contraction of the muscles of the hand and fingers, which came on in a barber at every attempt to use a razor.

A keirophobia as seen by me must be differentiated. This is a feeling of fear which came on every time the barber attempted to shave a man. An occupation neurosis also occurs in lapidaries (Stephan).

Sawyers' cramp (Poore) seems to be rather a craft palsy. An occupation neurosis of the labial muscles was seen by me in a horn-blower. As soon as he placed the mouthpiece to his lips a spasm came on in the orbicularis oris, so that the patient was unable to bring out a tune. It could not be determined whether it was a spastic or a paralytic condition.

Stammering and stuttering are closely allied to the occupation neuroses. A painful exhaustion of the laryngeal muscles in public speakers, etc., is called by B. Fränkel mogiphonia.

Occupation cramps are more rare in the lower limbs. A dancers' cramp, occurring in ballet dancers in the beginning of dancing when gliding forward upon the toes, which consists of painful tonic contractions of the thigh muscles, has been described. An occupation neurosis is said to occur in workers who use their feet in a treadmill.

A visual occupation neurosis may also be observed,—for instance, in continual use of a microscope, a spasm of the muscle of accommodation resulting.

Orbicularis spasms have been noticed in watchmakers (T. Cohn); there is also a watchmakers' spasm of the hands (Wilde).

A spasm of the ocular muscles occurring during military drills in fixing the eyes in attention has been described by Tranjen.

The "nystagmus of the mountaineers" might also be included here. I found this disorder in a violinist who was forced to read notes from a high stand with bad light.

These occupation neuroses must not be confused with the occupation palsies (professional pareses (page 341).

These pareses are due to excessive use of certain muscles, and are generally caused by a neuritis. Close examination should prevent any error, therefore, as the paralysis is persistent, of a degenerative character, and is often combined with sensory disturbances. Combinations of the occupation neuroses with a neuritis may, however, occur.

The treatment is similar in detail to that given under "Writers' Cramp."

TETANY (TETANILLA, IDIOPATHIC MUSCULAR SPASMS; ARTHROGRYPOSIS, ETC.).

This disease, first described by Steinheim and Dance, consists in *intermittent, bilateral, tonic,* mostly painful, spasms, occurring especially in *definite muscular groups* of the upper extremities, without, as a rule, any disturbance of consciousness accompanying them.

Appearing in some countries, as Sweden, and in certain districts, only rarely, in other places (*e.g.*, Vienna), it occurs in *epidemics* in certain months (March and April). Young men of the ages of sixteen to twenty-five years are particularly attacked, and these are mostly from the lower classes. It is also not rare in childhood; women are almost always affected during the time of pregnancy or lactation; even old age is not exempt, though it is rare after full maturity.

A number of facts indicate that it has a toxico-infectious origin: (1) its epidemic, endemic, and even familial occurrence; (2) its development after an *infectious disease;* (3) the part which some *poisons* play in the etiology (ergotin, alcohol, chloroform); I saw it occur after an injection of spermine, with symptoms of collapse, and in another case soon after the eating of crabs; the nephritic tetany perhaps also belongs here; (4) the relationship existing between tetany and *gastro-intestinal* disorders, to be discussed later; (5) the toxic products found in the stomach (Bouveret and Devic), and especially those found in the urine of tetany cases (Ewald and Albu).

Other facts do not, without further explanation, tend to indicate that a virus is in play, or appear to show that the spasms may occur from other causes. To these belongs above all others the fact that certain *occupations* seem to produce a noticeable predisposition. In three hundred and fourteen male patients (Frankl-Hochwart) one hundred and forty-one were shoemakers and forty-one tailors, while other occupations presented only single cases. The prevalence of tetany among shoemakers had been noticed previously, and had given rise to the idea that tetany was an occupation neurosis, a *cobblers' cramp.* It is my belief that the occupation is only an indirect cause, that the individual, through it, is brought in touch with an unknown poison, which in some way clings to the leather which he uses. Another observation which may be brought into harmony with the toxic theory is that *total extirpation of the thyroid gland* often produces tetany (N. Weiss and Billroth), while partial extirpation seldom has this influence. It is probable—yes, certain—that by the extirpation of the thyroid gland materials tend to accumulate in the organism which may be regarded as producing this disease, and the fact that the gland secretes a fluid which is antitoxic to certain toxic products of metabolism is confirmatory. Lately, the view that tetany is due to increased function of the thyroid gland, which may be excited by other causes, has been gaining in favor.

Healthy children rarely become sick with tetany; on the contrary, it is relatively frequent in those who suffer with gastro-intestinal disorders, especially *diarrhea*, also in *rhachitic* children (Ganghofner). Kassowitz goes too far in ascribing all cases to this factor. Tetany in children may occur in combination with laryngospasm (Loos, Escherich) and general convulsions. The opinion of Loos, however, that spasm of the vocal cords is a constant symptom of tetany, has not been confirmed (Hauser). Our knowledge of tetany in children and its relations with rhachitis and laryngospasm needs to be more closely studied. This disease may also appear in connection with osteomalacia (Blaziczek). Adults who suffer with gastro-intestinal disorders are also occasionally affected with tetany, severe forms having been observed in *gastric dilatation* (Kussmaul). It has not been settled whether the spasms result from reflex action or whether a toxic substance is in play; the circumstance that it has been observed several times immediately following the use of a *stomach-pump*, or the passing of a sound, speaks for the former. The observation that the spasms disappeared after the removal of intestinal worms—which observation, however, has not been accepted by all—confirms this. Gerhardt was able to produce the spasms in a case of tetany with dilatation of the stomach by slight percussion of the gastric region.

Exposure to cold, over-exertion, and emotional excitement are also considered exciting causes.

Symptoms.—The tonic muscular spasms of tetany do not come on suddenly and unexpectedly. As *prodromes*, paresthesia, pains in the limbs, and more rarely general disturbances, as headache, vertigo, and apathy, are observed.

The upper extremity is the most frequent and often the only part of the body affected by the spasms, especially the muscles moving the hand and fingers.

In typical cases, the *interossei* and other small muscles of the hand are involved so markedly that the hand assumes a characteristic position, often permitting the disease to be recognized at first glance. The basal phalanges are strongly flexed, while the digits are extended at the internodal articulations (Fig. 280), the fingers pressed apart, and the strongly adducted and apposed thumb is brought so far inward that it is completely covered by the fingers. The hand itself may be flexed on the wrist-joint, or the elbow may be flexed and adducted.

The hand does not always assume this *writers'* or *accoucheurs' position.* The fingers may be extended, the hand may be clenched, etc. The spasm does not confine itself to a few fingers in the majority of cases.

The *lower extremity* often takes part, especially the flexors of the foot and toes, which are found in a state of tonic contraction, so that the toes overlap or are flexed, and the foot is in a forced equinovarus position. The other muscles of the legs may also be affected.

Only in severe cases are the muscles of the *trunk* and the muscles of the *tongue, throat, respiration,* etc., involved. The ocular muscles may also be affected (Kunn). Nystagmus may be evoked by the spasms. Even the sphincter and ciliary muscles may take part.

An attack lasts a few minutes or hours, a day or days (up to ten). It may be so mild that the muscular contraction may easily be overcome, and in other cases so severe that the limb cannot be brought out of its forced position. Attempts to do so cause great pain. The muscular contractions in themselves also produce pain.

The spasm gradually lessens, though sensations of tension or other types of paresthesia may continue for some time afterwards. More rarely a paresis of the affected muscles exists for some time in the intervals.

The patient remains conscious, except in a few cases of intoxication tetany. It is said that disorders of consciousness are not uncommon in the tetany of childhood.

The *temperature* may be elevated slightly; a subnormal temperature has also been often noticed. The pulse-rate is generally accelerated during an attack.

The attacks may be separated from each other by intervals of hours, days, or weeks; as a rule, they follow each other very quickly, and sometimes only a slight relaxation of the muscles occurs between them. They may sometimes be excited by active movements (Bechterew, Schultze), mechanical stimulation, etc.

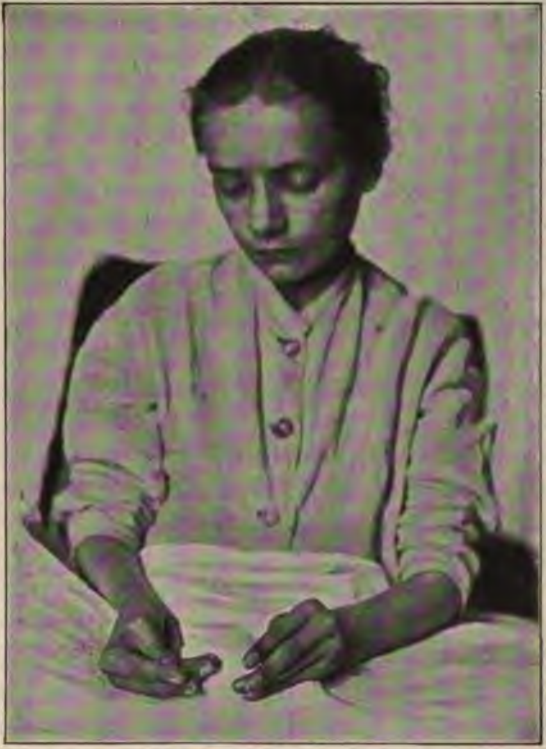
Although the spasm is the important symptom of tetany, we find in addition a number of characteristic signs. To these belong—

1. *Trousseau's Phenomenon.*—Trousseau found that pressure upon the affected limb will bring forth an attack, "the nerve-trunks or the

blood-vessels being compressed in such a manner that the arterial or venous circulation is impeded."

In a majority of cases we may produce an attack during the intervals by pressure exerted in the region of the internal bicipital sulcus. Sometimes it can be evoked in one-half a minute, on other occasions only after three to four minutes' pressure. It is believed that the anemia produced by compression of the arteries is to blame. The fact that in some cases in regions in which no peripheral arteries can be found—for example, the wrist-joint—the spasm can be produced by pressure, is opposed to this view. V. Frankl-Hochwart has shown experimentally that it is the nerve-compression which—probably reflexly—excites the spasm. Trousseau's sign is not observed in any other disease of the nervous system; absence of it, however, does not indicate the disappearance of the tetany.

FIG. 280.



A case of tetany during an attack.

2. *Increase of the Mechanical Excitability of the Motor Nerves* (*Chvostek's Sign*).—Slight percussion of the motor and mixed nerves

will produce lively contractions of the muscles innervated by them. This, as a rule, appears most prominently in the *facial* nerve, and gives rise to the so-called facial phenomenon,—pronounced twitchings in the facial muscles, produced by drawing the handle of a percussion hammer over the branches of the major anserinus. Increase of the mechanical excitability of the nerves is also found in tuberculosis, simple gastric dilatation, and also in other nervous diseases, but is rarely so marked as in tetany. This symptom is, on the other hand, not very pronounced in tetany of children (Schlesinger).

3. *Increase of the Electric Excitability of the Motor Nerves* (*Erb's Sign*).—This is found in almost all cases. A very weak current will produce the *Ca.C.C.*, and a very slight increase of the current strength will produce *Ca.C.Te.* (the ulnar nerve reacted in one of my cases to 0.1 *M.A.* with *Ca.C.C.*; to 0.5 with *Ca.C.Te.*); and *A.O.Te.* and sometimes even *C.O.Te.* may be produced.

The increase of the electric excitability is less marked with the faradic current.

4. *Increase of the Mechanical and Electric Excitability of Sensory Nerves* (*Hoffmann's Sign*).—In many cases an increased excitability of the sensory nerves may be detected. Slight pressure on the supraorbital, ulnar, auricularis magnus, saphenous major, and other sensory nerves may produce a radiating sensation,—a paresthesia of the region innervated by the nerve,—while a healthy person experiences only a local sensation, if any, from such pressure.

The sensation experienced by a healthy person whenever the ulnar nerve is struck or subjected to pressure at the elbow-joint, is felt with very slight stimulations. This symptom is, however, by no means pathognomonic of tetany. We have very little information concerning an increased electric excitability of the sensory nerves.

In healthy persons, weak galvanic currents produce first, a cathode-closure sensation, which soon passes into a cathode-closure persistent sensation, then follows the anode-closure sensation, and the anode-closure persistent sensation. Finally, in increase of the strength of the current we get not only local but also radiating sensations. In tetany these sensations are produced by much weaker currents, and the intervals between local and radiating sensations are too slight to be separated.

An increase of the electric excitability of the *auditory* nerve has also been observed (Chvostek, Jr.).

Bechterew has lately called attention to the fact that repeated stimulation produces a progressive increase of the mechanical and electric excitability. He calls this the reaction of excitation.

Inconstant Symptoms.—Hyperidrosis (particularly after an attack), polyuria, albuminuria, and glycosuria have occasionally been observed, also edema, urticaria, herpes zoster, pigmentation, and loss of hair and falling out of the nails.

Paralysis of certain muscles,—particularly of the gluteal, pelvic, and lumbar muscles,—atrophy, and anesthesia are rare symptoms.

The reflexes vary, being decreased, normal, or increased ; Westphal's symptom has been observed several times. *Mydriasis, pupillary rigidity,* optic neuritis (?), cataract (Magnus), and psychic disorders are rare symptoms. In a case of strumiprivous tetany, Westphal's symptom and pupillary rigidity were found. In another case, Hoffmann found a myotonic reaction. Psychic disorders similar to hallucinatory confusion were observed by v. Frankl-Hochwart in three cases. Schultze has made similar observations. It is not so uncommon to find epileptic conditions combined with tetany ; it may also combine itself with myxedema, exophthalmic goitre, and other diseases.

Diagnosis.—The spasms are so characteristic that the disease may be recognized with little difficulty. Tonic muscular contractions of a similar nature occur in hysteria,—*i.e.,* hysteric pseudotetany,—but are mostly unilateral, and Trousseau's and Erb's signs are absent. The attempt of French authors to make tetany an hysteric disease has not succeeded. If the tetany extends to the muscles of the trunk and jaw and to the respiratory muscles, a superficial examination may tend to confuse it with tetanus, though the development of the spasm, its onset in the muscles of the hand, the late appearance of the trismus, and the intermittent character of tetany attacks in contrast to the more continuous spasm of tetanus should prevent such errors.

There is a *tetany without spasms,* in which the patients complain of paresthesia, especially in the hands, and in addition we find Chvostek's and Erb's symptoms. These *tetanoid* conditions may change into true tetany.

Latent tetany also occurs in childhood (Hauser, Escherich).

Tonic spasms involving more or less the whole musculature of the body, and occurring with *albuminuria,* have been observed several times (Kussmaul, Kast). Their nature is still unsettled.

Kjelberg and Escherich observed in children tonic spasms lasting for weeks and months with eventual recovery. The condition had a superficial similarity to tetanus (pseudotetanus).

Henoch described similar conditions under the name of *idiopathic contractures ;* he observed them in rhachitis, dyspepsia, eclampsia, etc., and is not inclined to identify them with tetany.

It is doubtful that tetany limits itself to a few muscles (*e.g.,* a tetany of the diaphragm with the symptoms of asthma) ; Neusser has observed such a case.

Pathologic Anatomy.—The observations made so far (Tonnelé, Schultze, Berger, Kohts, Weiss, Schlesinger, and others) have not led to a unanimous opinion. The autopsy has often been negative. Hyperemia and circumscribed hemorrhages, cloudy swelling of the ganglion cells, softening, and even a poliomyelitic alteration of the spinal cord, have been found. Although these results indicate the anterior gray matter of the spinal cord as the starting-point for the disease, they are so few and antagonistic that they are of little value.

Course and Prognosis.—The disease may run its course in a few days, with one or a few attacks. It often lasts weeks and months. There is also an acute relapsing (Jaksch) and a chronic form which lasts with remissions for years. Some individuals are troubled every winter with tetany.

The prognosis in regard to life is good, except in those cases of tetany which occur with *gastric dilatation*, also in those which are produced by *extirpation of a goitre*, in which it may be combined with myxedema. The disease may, however, have a good ending in these cases also. Suckling babes and children often die from the original disease (intestinal catarrh), while otherwise healthy children, as a rule, recover. It may have a fatal result if the respiratory muscles are attacked.

Recovery occurs in a majority of the cases. Cases occurring after infectious and intoxicating conditions are especially characterized by a rapid, mild course. I have seen such cases who had only a single attack. In pregnant women, it may last till delivery is over or disappear earlier; the spasms occurring during lactation generally disappear with the completion of this function.

Treatment.—As a prophylactic measure, only partial resection of the thyroid should be undertaken, instead of total extirpation. If the disease is due to the influence of cold, a *diaphoretic* treatment must be instituted.

The underlying causal condition must in all cases be removed. In the treatment of gastro-intestinal disorders, the use of the stomach-pump should be avoided if tetany is present.

The intensity of the spasms may be lessened by the use of *potassium bromide*, *morphine*, and *chloral hydrate*. *Hyoscin* and *curare* have been prescribed in stubborn cases. Hoche claims to have shortened the attacks in one case with curarine (0.0003 to 0.0006). Kassowitz claims to cure tetany of children with phosphorus. Faradic currents should never be given; a stabile application of a mild galvanic current may be tried.

Warm baths and *wet packs* are often of benefit. Trousseau recommended the application of the ice-bag to the vertebral column, also bloodletting.

The future alone will tell us of what value in the treatment of tetany strumipriva the results obtained by transplantation of the thyroid tissue in animals, whose thyroids have been extirpated, and those in myxedema, will be. I have seen one case recover, it having been under observation two years now. Mannaberg reports only failures.

We have seen recovery occur from thyroid-feeding, as have others. Some report bad results (Mannaberg). In one case, pilocarpin was reported to have cured the tetany. Lactation must be stopped. Weak children should be well nourished and be given iron, quinine, or cod-liver oil.

CHOREA MINOR (ST. VITUS'S DANCE).

Under the name chorea, are included many different disease conditions. They are all characterized by muscular contractions and involuntary movements of a complicated nature. As these disease forms are, excluding this symptom, of a heterogeneous nature, a sharp differentiation between them is demanded. Chorea minor,[1] also called Sydenham's chorea, is particularly a disease of childhood and youth. It occurs very rarely before the fourth year, and in a vast majority of cases is found between the fifth and fifteenth years of life. Girls are affected three times as frequently as are boys. From the fifteenth to the twenty-fifth year of age, the cases are almost entirely females. From this period it becomes more rare; no time of life is, however, entirely exempt. The designation *senile chorea* in itself denotes that it may occur in old age. *Tender, anemic, irritable individuals* are especially predisposed; a *neurotic make-up* increases the predisposition. Thus it occurs that chorea and hysteria often combine; we do not, however, refer to those cases of *chorea hysterica*, in which hysteric muscular contractions occur. The presence of nervous diseases in the family of choreic children may be established in not a small percentage of cases.

The disease often develops without any noticeable cause. An emotional shock, particularly a fright, has often been blamed. The terrifying moment is, however, sometimes so unimportant that only a marked increase in excitability would explain the effect; it may also occasionally be determined that the germs of the disease have been present before the emotional outburst came on.

Mental excitement is a more important etiologic factor in the chorea of adults than in that of children. It is especially girls of the age of from sixteen to twenty-two years who are the victims of chorea; in the cases of this age observed by me I could generally trace the attack to an emotional shock.

---

[1] Chorea major or magna is not an independent disease but an hysteric condition.

Chorea may arise from *simulation;* endemics of this disease have been observed in boarding-schools, etc., those affected being, however, mostly hysteric. Probably the chorea acquired from imitation is not a true chorea, but an hysteric disease.

The relations of chorea to *pregnancy* are definite. In previously healthy individuals, or in those who have had chorea once before in childhood, the disease appears in the first months of pregnancy, especially in from the third to the fifth month. They are generally *young primiparæ,* relatively often unmarried pregnant women, the pregnancy appearing to increase the disposition, and some other factor, particularly mental excitement, being the exciting cause. The relationship to pregnancy is so close that the disease ends with the cessation of pregnancy, be it that pregnancy proceeds *ad terminum* or is broken by abortion or miscarriage. The chorea often recurs in later pregnancies. It is rare for the chorea to come on only during the *puerperium.* Some observations seem to indicate that *traumata* may also produce chorea. The etiologic importance of masturbation is doubtful.

The relations existing between *chorea, articular rheumatism,* and *endocarditis* are variable. Even though the opinions of different physicians vary, it is an established fact that chorea occurs extraordinarily often after acute articular rheumatism. In many cases the latter disease produces an endocarditis, and the St. Vitus's dance occurs after its onset. It also happens that the endocarditis is only discovered during the course of the chorea; or that the rheumatism only occurs during its course; or we find an old cardiac disturbance in a patient suffering from chorea. Diseases of the heart occur more often in chorea of youth and pregnancy than in that of childhood.

Many theories have been advanced to account for the disorder. Some observations seemed to indicate that embolic material passing from the heart to the brain and causing there occlusion of the small vessels and circumscribed areas of softening produced these motor symptoms of irritation. Rheumatism, however, often evokes chorea without the connecting link of endocarditis, and embolisms have been detected in only a small number of cases.

Some imagine that the infectious process itself produces the thrombosis of the cerebral capillaries. It has also been thought that the cardiac disease produced the chorea in a *reflex* manner. Finally, others claim that a micro-organism or virus is the cause of the chorea as well as the arthritis and endocarditis.

A few authors (Laufenauer, Triboulet, Bechterew, and others) always refer it to an infectious cause, and look upon the other factors as exciting causes. Positive evidence of micro-organisms in the brain of choreics has been found in but few cases (results of Richter, Berkeley, Dana, H. Meyer, Pianese). Pianese found a diplococcus and a diplobacillus, cultures of which produced an experimental chorea.

In only a few cases has chorea been observed after scarlet fever, measles, and typhoid.

Comparing the different etiologic moments we must come to the conclusion that chorea may be produced by different poisons. That age is especially in danger in which the motor inhibitory apparatus is not completely developed, in which mental excitement uninhibited is transformed into motor actions. We often observe in children, young girls, and women under the influence of worry and other emotions, a motor restlessness which is very similar to the picture of chorea. It is possible that individual increase of this predisposition may be an exciting cause of chorea.

The disease is then produced by a severe emotional disturbance, or by pregnancy (reflex action?), or, more commonly, through the action of a poison which exists in the body of a person affected with articular rheumatism or endocarditis or which enters from without. Epilepsy furnishes a certain analogy in being inherited or produced by emotional excitement, infection, and intoxication.

Symptomatology.—The disease generally develops insidiously. The children become restless, awkward, and careless. The teacher complains that the child cannot sit still, or that his writing has become slovenly. It is not rare for the child to be considered naughty. Soon, however, a diseased condition becomes pronounced.

During the physician's examination the *motor restlessness* is particularly noticeable. The child does not sit or stand still, but is in continual motion. The arm is abducted and adducted, or rotated; the shoulders raised; the hand extended, the fingers separated, to become immediately flexed or extended again; the trunk is turned or rotated from one side to another; the head thrown to one side, etc. The gait is disturbed in severe cases; the contractions of the legs may be so marked as to prevent the patient from standing. The above movements follow each other in constant variation, so that different muscle regions and the muscles of different extremities are brought into action at the same time. It is characteristic of chorea that the involuntary movements do not confine themselves to one muscle-group and occur rhythmically in it, but that they appear now here, now there, and in irregular order involving the different muscle-groups. Single motor acts do not correspond so much to the type of twitchings as to the type of movements; they resemble voluntary movements, but, unlike them, are *purposeless and vary constantly in form and direction.* Rapid twitchings—*e.g.*, in the facial musculature—are often observed in addition to the combined movements. Laughing and crying are very labile and easily excited.

The *upper extremities* are more often affected than other regions, next the face and trunk, lastly the lower extremities. The *tongue* and other articulatory organs often take part in the twitchings. The tongue is thrown around in the mouth or protrudes through the teeth, causing a certain amount of disturbance of speech. The words are tossed out, suddenly broken off or indistinct, or separated by irregular respiratory movements. Gurgling and smacking noises are also produced by the action of the glossal musculature. The speech may be disturbed so much that the patient may not utter a word for days or weeks.

The protrusion of the tongue occurs hastily and it is retracted just as quickly. The involvement of the muscles of the lips, tongue, palate, etc., may cause difficulty in eating. The *respiratory* muscles are generally affected, especially the diaphragm; irregular spasmodic breathing occurs.

The muscles of *phonation* are rarely involved. In many cases the ocular muscles are also affected; the patient does not fix his eyes, but rolls them around from one side to the other. A transient *strabismus* may also be produced by the twitchings.

The disorder generally commences *in one arm,* to radiate to the other, or to the leg of the same side. In many cases the chorea confines itself to one side of the body (*hemichorea*), or generalizes itself only later in its course.

Voluntary movements generally increase the choreic twitchings. The former are only affected when the involuntary ones follow them, combine with them, and modify them. The patient, therefore, seeks to execute active movements as quickly as possible, and seeks to use those moments in which the choreic movements have been repressed. His movements, therefore, become rapid and brisk, and the patient is unable to finish any work that requires continued co-ordinated movements. In mild cases, however, the unwilled movements may not hinder the voluntary movements.

Emotional excitement has the greatest influence upon the muscular unrest. Conversation with the physician, even the feeling of being observed, may increase it noticeably. Some patients, however, may control themselves before others; but in them the restlessness may be brought out by the examination, or by asking them to recite a poem, etc. Mental rest is, therefore, of good influence, and the fact that the twitchings generally cease during *sleep* confirms this. In rare cases, however, the motor restlessness is most pronounced during sleep, and almost disappears during the day (*chorea nocturna*).

The intensity of the twitchings varies in different cases. They may be so slight as to be scarcely noticeable, or so severe that the patient jumps here and there, injures himself, bites his tongue, cannot eat, and from his *muscular insanity* (*folie musculaire*) presents a horrible appearance.

Brissaud and Patry speak of a "variable or polymorphous chorea," which occurs particularly in degenerates, and is characterized by inconstancy of the twitchings. They may recede for days, and vary exceedingly in intensity and character.

The involuntary movements form the principal, and at times the only, symptom of this disease. In a majority of cases, however, the *psyche* is affected,—irritability, depression, peevishness, and weakness of the memory combine with the motor restlessness. These psychic defects increase—rarely in chorea of childhood, often in adults—until a real *psychosis* is present, *hallucinatory delirium*, with severe maniacal excitement and mental confusion, or, more rarely, an *acute paranoia*.

The *mania* generally develops at the height of the chorea, lasts only a few weeks, and then depression, apathy, or some mental disorder, characterized by hallucinations, ideas of persecution, self-reproach, etc., takes its place. The mania may, however, last a long time. The most severe forms occur in the chorea of pregnancy.

Paralytic symptoms do not belong to the clinical picture of chorea, the motor strength being unimpaired ; there are, however, a few cases in which the disease begins with a sort of *paresis* or *pseudoparesis*, and to a certain extent hides itself under a paralytic condition (Todd, West, Charcot). It appears to the family as if the child moves one arm less than the other one, and, finally, does not move it at all. This motor disturbance may involve both arms, arm and leg of one side, or even the whole body, so that the patient lies as if paralyzed (limp chorea, paralytic chorea). It is, however, able to move its limbs upon command, though without energy, and for only a short time.

On closer examination, we find transient twitchings which reveal the choreic nature of the disease. They are so slight that they must be sought after, though they may appear prominently in the non-paralyzed muscles. In its further course, the twitchings become more marked and the pseudoparesis gradually disappears. Charcot insisted, however, that the twitchings may cease during the chorea and a kind of paralysis take their place.

The muscles retain their normal volume and their normal electric excitability.

The deep reflexes are normal, though they were absent in a case of paralytic chorea described by Gumpertz. The sensibility is not altered in typical cases, but only in hysteric chorea, though a concentric contraction of the visual field appears to occur in simple chorea.

The ophthalmoscopic examination does not reveal anything pathologic. Optic neuritis has never been observed by me, although it is said to have occurred in a few cases. The pupils are often dilated, but react promptly to light. The pulse is often accelerated, rarely arrthymical. The sphincters functionate normally.

The general constitution is disturbed only when the forcible muscular movements prevent the ingestion of food and produce exhaustion. Increased temperature is found in severe cases. In fatal cases a temperature as high as 109° F. has been observed.

Complications.—We have previously referred to the combination of chorea with the psychoses. It is also often associated with the *neuroses.* It shows a special predilection for combining with *hysteria.* Chorea may develop in an hysteric individual, and remain independently of it, or it may cause hysteria in predisposed persons; or an hysteric chorea may be present,—*i.e.*, a neurosis of motility upon an hysteric basis which, notwithstanding a close relationship with true chorea, differs from it in many important points, particularly in the sudden onset after some emotional shock, its development through imitation, and especially by the character of the twitchings. They consist in rhythmic, sometimes forcible, contractions of the extremities. They are systematic movements recurring in a stereotyped manner, may last for days, weeks, or months, to disappear suddenly after a spasm, an emotion, or with the onset of the menses, etc.

Combinations of *epilepsy* and chorea occur more rarely. Congenital imbecility may also be accompanied by a stabile chorea.

The most important of all complications of chorea are *endocarditis* and a *vitium cordis.* Mitral insufficiency is the most common disturbance. In chorea an accidental cardiac murmur should not be made the basis of a diagnosis of an organic disease of the heart. As the patient is often anemic, it is not rare to hear a systolic blowing over the mitral and pulmonary valves, which is simply an *anemic murmur.*

The endocarditis occurring in chorea is generally of a mild nature, and may disappear without doing any harm. It sometimes occurs that in the course of chorea an embolus from the diseased heart reaches the brain and produces paralytic symptoms.

The articular rheumatism developing during chorea is generally of but slight severity. The patients complain at times of pains in the limbs, without there being any local alterations noticeable. These pains naturally should not be referred to rheumatism.

Course, Duration, and Progress.—Chorea has an average duration of two or three months, but it often lasts much longer,—from six months to a year. Only in rare cases does it extend over a longer interval or assume the character of an incurable disease. There are also cases so mild that they recover in a few weeks. The prognosis is good: complete recovery is the rule. Life is only slightly endangered. In the chorea of childhood there are about three—at the most, five—deaths in a hundred cases. These were particularly severe forms in which the muscular contractions had disturbed sleep and nutrition and produced an exhaustion which caused death from itself or through the agency of a *fatty degeneration of the heart*. Injuries, to which the patient becomes liable on account of his movements, may also help in bringing on death, on account of the difficulty of treating them.

The prognosis in regard to life itself is not rendered worse on account of an endocarditis, as it only rarely causes death. It is true, however, that almost all choreics that have died were cardiopaths. In adults a fatal termination is not so rare. Death rarely results from relapses.

Chorea of pregnancy has a more serious prognosis. About twenty-five per cent. of such cases die. This is partly due to the twitchings often becoming very severe in such cases, to the frequent combination of the psychoses and severe forms of endocarditis with this form of chorea, and to the cachectic condition caused by abortion, or a spontaneously occurring or artificially produced miscarriage, or by a birth coming on after a normal pregnancy. If these complications are absent, recovery is here also the rule, and generally takes place with the cessation of pregnancy, lasting longer than delivery only in a few cases. The same is true of the psychic alterations, which rarely persist after the termination of labor. The prognosis as to life for this form of chorea seems to be more favorable in cases in which chorea has occurred in childhood.

Rapid emaciation, delirium, rise of temperature, etc., are dangerous symptoms when occurring in the course of a chorea.

The older a patient is the longer is the chorea liable to last. It has seemed to me that in mentally weak children the chorea drags along for a long time. On the other hand, hopes for recovery must not be given up on account of the long duration of the disease. In a woman who had chorea from her seventh to her twenty-fourth year it disappeared with her first pregnancy. If it develops late in life, the probability is that it will become permanent. Chorea is a disease which is inclined to disappear, to recur later on. The intervals between the attacks are generally about twelve months, though there is no regularity. Some cases occur in which the attacks come on so soon after one another that we speak of a *chronic intermittent chorea.*

We also recognize a *chronic perennial* form, which may last for years or for a lifetime. This type is rarely observed in children; at least it is very rare for a chorea of childhood to become stationary. Chorea of adults may, however, become a "chorea (adultorum) permanens." We do not refer to the hereditary form here, which will be discussed later.

The ordinary chorea minor may come on in the aged and run its typical course; as a rule, however, senile chorea is a permanent form.

Differential Diagnosis.—The diagnosis is extremely easy in most cases. Some difficulty may be experienced in those cases in which the disease commences with a kind of paralytic weakness of the arms. Closer examination will, however, reveal the twitchings; the psychic condition will also support the diagnosis. In those cases in which the chorea has existed since early childhood, it may be mistaken for the choreic-athetotic form of cerebral infantile palsy (which see). This may extend over the entire body, and thereby render the choreic factor so prominent as to conceal the paralysis. Close examination will, however, reveal some muscular rigidity, particulary in the legs, associative movements, and the athetoid character of the twitchings. In addition, a congenital or permanent form of chorea coming on in childhood is very rare, and should always give rise to the suspicion of an organic cerebral disease as the basis of the motor symptoms of irritation.

Chorea sometimes localizes itself in the legs, and produces a disorder of gait similar to ataxia. In one case of this kind, in a three-year-old boy, I thought it was a spinal affection until the involuntary movements were observed in rest. The disease had come on after a fall, and disappeared after a few months.

Chorea is often mistaken for general tic; the differential points have been described under this disease. In a few cases we find a *partial chorea*, limited to the musculature of speech or of the eyes, or to the muscles of the lips, tongue, pharynx, or larynx. These are severe, although limited in extent, and often very stubborn.

Pathologic Anatomy.—Only severe, complicated cases, as a rule, come to an autopsy. As a cause of death we find in most cases an endocarditis, valvular disease, or a fatty degeneration of the cardiac muscle. Although the cerebral examination was generally negative, the most variable alterations have been noticed, especially hyperemia of the brain, hemorrhages, foci of inflammation and of softening, especially in the central ganglia, arterial disease with thrombosis, inflammatory alterations of the cerebral meninges, hematoma of the dura mater, etc. (Meynert, Dickinson, Ogle, Anton, Dana, Nauwerck, Macleod, Kroemer, and others). Emboli of the larger arteries, with necrobiosis or stopping up of smaller vessels, and capillary emboli of the central ganglia have been noticed occasionally.

The *chorea corpuscles*, found in the lenticular nucleus and referred to chorea, were later found in the brain of individuals who died from other causes (Wollenberg). Some authors claim to have found minute alterations in the ganglion cells. Pathologic alterations have also been noticed in the spinal cord (Hutchinson, Clark, and others). But they are inconstant or variable changes, and have been found in only the severe and complicated cases, so that they throw no light upon the nature of the disease. Gowers considers them to be secondary alterations. Bechterew is inclined to believe that the infectious agents do not, as a rule, produce gross alterations in the nervous system, but may occasionally do so.

Chorea is, without doubt, a disease with an unknown anatomic basis; and it is certain that gross anatomic lesions are not present, and that in typical cases either no material disease is present or only minute alterations which are capable of retrocession.

On the other hand, it is certain that choreic twitchings may be present in the symptomatology of organic cerebral diseases. (See Hemiplegic Chorea, page 440.) There is no doubt that chorea is a cerebral disease.

Treatment.—The enormous influence which mental excitement has upon choreic movements and the increased excitability which is found in choreics render evident the necessity for avoidance of all emotion and excitement. The child should not be allowed to go to school the stay at home must be one of *isolation* from its playmates, from its sisters and brothers; and only the mother or a nurse should form the company for the child. The child should be amused by some occupation without over-exerting or exciting it. Threats and warnings should be avoided; it is an error to believe that the patient can voluntarily repress the chorea. Wherever threats and punishment produced a result, an imitation-chorea (simulation) had probably been present. The child should be placed in a large, airy room; in mild cases it may be allowed to go out into the open air. An easily assimilated, nutritious diet is in place. Coffee, tea, and alcohol should be forbidden, while large quantities of milk should be given.

If the chorea is severe, the child should restrict its active movements. Rest in bed is then necessary. It is important that a choreic individual sleep long and soundly. If the sleep is poor, *hypnotics* should be given. If the contractions are so severe as to render injuries possible, the child should be placed in a padded corner on the floor, and the walls should be covered with mattresses or pillows. Only where a psychosis is present is it necessary to transfer the patient to an institution. When the house arrangements are inferior, treatment should be carried on in a hospital.

Drugs.—Arsenic should be used in every case, as its therapeutic value is considerable. About four drops of Fowler's solution should be given to a five- to ten-year-old child and the dose gradually increased to eight or ten drops. Arsenous acid is more reliable and more powerful, and may also be given in combination with iron. As soon as the arsenic produces indigestion, conjunctivitis, herpes, or other symptoms of intoxication, it should be discontinued. The *bromides* may do good and *chloral hydrate* may be of benefit in severe cases.

Chloroform should only be used in the severest cases, remembering that it is in just these cases that the heart is weakened. *Subcutaneous injections of morphine* have been of good service in some of my very severe cases. Zinc salts, conium, hyoscin, cannabis indica, etc., are probably valueless in all cases. *Antipyrine* has sometimes a beneficial action; *physostigmine* has also been recommended. Exalgin, salol, salophen, etc., have been recommended in recent years.

Mild hydrotherapeutic measures, simple washing, or partial rubbings with cold water or warm half-baths should be instituted in every case. Electric treatment is of doubtful benefit, though galvanization and electric baths are recommended by some.

For the treatment of the chorea of adults, the same measures should be undertaken. Isolation is even more important than in infantile cases, on account of the psychic condition.

Chorea of pregnancy demands special attention. In the use of remedies the fetus must be considered, and arsenic, etc., must, therefore, be used with caution. The artificial termination of gestation may sometimes be a vital necessity. It should only be undertaken, in cases where the child is not old enough to be born, where the severity of the contractions, the exhaustion, cardiac lesion, nephritis, psychosis, etc., endanger life.

In convalescence, gymnastics are advisable, also a sojourn in the country, mountains, or at the sea.

OTHER FORMS OF CHOREA.

HEREDITARY CHOREA (CHRONIC PROGRESSIVE CHOREA, HUNTINGTON'S DISEASE).—This is a disease *sui generis*, and must be sharply separated from chorea minor.

It is, on the whole, a rare disease, which is passed on from generation to generation. It may skip a generation, or epilepsy and hysteria may occur in its place in this generation. It appears that if a member of the family is spared, his descendants are liable to be free from the disease.

Men and women are equally affected. It commences, generally, between the thirtieth and fortieth years of life, it may, however, commence sooner or later. An exciting cause is entirely absent, or the first phenomenon follows some emotion.

The cardinal symptom of this disease is the motor phenomena. At first of slight intensity, and limited to a few regions (face, upper extremity, etc.), they gradually increase in intensity and extent, and finally embrace the whole voluntary muscular system.

In most cases, however, the ocular muscles do not take part.

The picture, in general, corresponds to that of chorea minor; it is a varying picture of unwilled, purposeless movements in the different muscle-groups. They produce an almost unbroken series of *grimaces and gesticulations,* hinder speech, and occasionally hamper respiration. They generally cease during sleep, and are increased by mental excitement. All observers, however, have noticed that the patient, by will-power or by the execution of voluntary movements, may repress the choreic twitchings for a certain time. He is thus able to grasp an object, to thread a needle, to write, or to eat—all these acts do not increase the involuntary movements, but inhibit them. This control is, however, only temporary. This repression of the active extremity by will-power is often bought only at the expense of an increased unrest of the other muscles. The movements are also not continuous, and in the intervals the chorea asserts itself.

The patient can walk until the later stages of the disease, though the gait is peculiar. It is erratic, the trunk rocks, or the upper body is bent forward, or the legs move as in dancing, or the patient suddenly stops after taking a few strides—all of which is done rapidly and with constant variation.

The motor strength remains intact almost throughout the whole course of the disease, though in a few cases a paralysis (hemiplegic type) occurred later on. The sensibility and special senses are not affected. The deep reflexes are generally somewhat increased. The internal organs are normal.

Mental weakness is almost always present and progresses slowly, ending frequently in idiocy. Depression is present, leading in many cases to suicide. Increased irritability is often observed, which may increase to severe excitability; it is not rarely succeeded in the later stage by apathy. The mental enfeeblement generally comes on years after the onset of the motor phenomena, but may precede them.

The prognosis is very bad. It is an incurable disease. The individuals affected finally become bedridden, and die from some intercurrent malady, from cachexia, or in a comatose condition. It lasts from ten to thirty years.

Diagnosis.—In order to diagnose it from other forms of chorea it is necessary to find *direct heredity* in play. This does not occur in simple chorea, or is rare, and then only in the ascendants, not in the whole generation.

If the hereditary nature cannot be determined, a diagnosis from simple chorea cannot always be made at first. The further course, however, soon shows its progressive character, the mental enfeeblement, and the incurable nature of the disease.

Pathologic Anatomy.—Meningeal alterations, consisting of cloudiness, external hydrocephalus, and hemorrhagic pachymeningitis, have repeatedly been observed, though they are merely accidental discoveries, and not the causes of the disease. In two cases which I examined (with Hoppe) I found *disseminated miliary encephalitic foci*, especially in the cortex of the motor region. Similar observations have been made by Greppin (before us), Kalisher and Kronthal, Facklam, and others, though in the cases of the last-mentioned a diffuse meningomyelitis with secondary atrophy of the cortex was present. Binswanger claims that the cerebral findings in a case examined by him did not differ materially from those of paralytic dementia.

Whether the findings given were the anatomic basis of hereditary chorea is another question.

The treatment is a symptomatic one. Arsenic is rarely of value, although results have been claimed for it in one or two cases.

I saw, in company with Remak, a very peculiar form of hereditary chorea. Two children (sons) of a mother affected with permanent hemichorea became ill in their eighth year of a chronic progressive chorea, which began in the lower limbs and which in one resembled an athetosis of one leg. This gradually extended to other muscles until in a year the typical picture of a general chorea was present, the legs being affected the most. One patient cannot walk at all; the other walks with difficulty, backward better than forward.

ELECTRIC CHOREA.—Under this name are included various and, to some extent, obscure disease conditions. To these belongs, first, a disease observed in upper Italy and described by Dubini. It may occur at any age. Following a sensation of pain in the head, neck, or spinal cord, we find short, rapid spasms similar to those produced by electricity. They occur in one arm or half of the face, extend to the leg of the same side, and finally to the opposite side. Epileptiform attacks confined to one half of the body also occur. In its further course paralytic symptoms develop, first in the extremity in which the spasms commenced, afterwards spreading to the other limbs. It is combined with atrophy and disturbances of the electrical excitability. The skin is hypersensitive, the temperature may be increased, and the patient suffers much pain. In days, weeks, or months death results from heart failure or coma. Only a few cases have recovered.

Bergeron describes a disease as electric chorea which he has seen in children from seven to fourteen years of age, especially in anemic and irritable ones. The most important or, we might say, the only symptom is severe spasms occurring in impulses. The head is thrown here and there, the shoulders raised, the upper arm abducted, the lower arm flexed, etc. The movements are rhythmical, and may be limited or generalized in extent. They disappear in sleep and cannot be controlled by the will. All other functions are normal. The prognosis is good. Recovery ensues after some days or weeks from the use of arsenic, cold douches, or an emetic. This disease has been thought to be due to gastric irritation. It would be difficult to distinguish it from hysteric chorea.

Henoch has described a form of children's chorea as electric chorea, which is distinguished from ordinary chorea by the lightning-like character of the contractions. Some consider this disease of Henoch to be identical with myoclonus. The contractions attack particularly the nuchal and shoulder muscles, and occur at intervals of from two to five minutes.

All these diseases differ widely, and the name electric chorea should therefore be dropped.

PARALYSIS AGITANS; SHAKING PALSY (PARKINSON'S DISEASE).

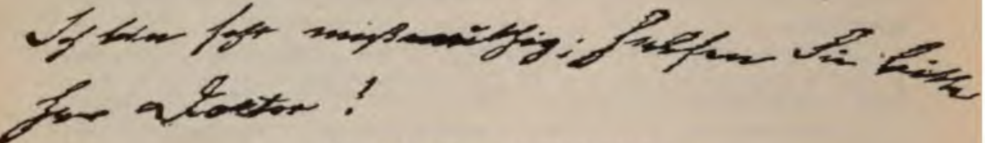
This is a disease of the aged. It rarely commences before the fortieth year, though it may occur earlier. I observed it in a thirty-two-year-old man and Lannois even in a twelve-year-old child. The *cause* is generally obscure. Persistent worry has been occasionally and a severe fright often blamed. Injuries of the head, the trunk, or an extremity, particularly crushing and laceration of the nerves, may be the cause of the disease, which in such cases commences in the injured part of the body. Hereditary influences do not play a prominent part, though present in some cases. Its relationship to acute infectious diseases is a doubtful one. Syphilis had been present previously in four of my cases, and in two of them the symptoms of the disease occurred remarkably early. An antisyphilitic treatment, however, was useless.

Symptomatology.—The disease presents the following characteristic symptoms: (1) *tremor;* (2) *continued muscular rigidity,* which produces a *peculiar position of the head, trunk, and extremities;* (3) *retardation of and difficulty in active movements;* (4) *a peculiar modification of the gait.*

The tremor occurs particularly in the extremities, especially the upper, and of these more particularly the peripheral parts,—hands and fingers. It consists of *rhythmic* oscillations, four or five in a second, rarely more, and which consist in flexion, extension, abduction, and adduction of the hands and fingers. The excursions are generally of small amplitude,—*e.g.*, the thumb and index-finger are rubbed against each other, as in crumbling bread, rolling a pill, etc. The tremulous movements of the hands are of larger amplitude and may at times increase to a true shaking.

A cardinal peculiarity of this tremor is its contin[illegible] when the patient is at rest. The tremor is present whether the patient lies down or stands up, lifts the hands or allows them to hang by his side, etc. Spontaneous remissions may occur; the tremor may stop for a time or pass to other muscles, but it is not first evoked by active movements. I will discuss later a few variations from this rule.

FIG. 281.



Ich bin sehr mißmuthig; helfen Sie bitte Herr Doctor!

Handwriting of a case of paralysis agitans.

The tremor is a stereotyped one: the same movement, with the same amplitude and rate of oscillation, occurs continually. A decrease or increase in intensity is observed occasionally, but the oscillations are about equal for many seconds, even minutes.

Active movements temporarily silence the tremor. It will stop momentarily if the patient clasps your hand or shuts his, etc. It continues or is increased in forced, active, or in continued movements, so that the handwriting reveals the tremor.

The tremor may also be increased by active movements when they are accompanied by excitement,—for example, in clinical demonstrations.

The inhibitory influence of active movements is recognized by the patients, so that they grasp some near object or alter the position of their limbs and thereby repress the tremor; it is not uncommon for them to feel better when walking than when quietly sitting or lying down.

Passive motion also exercises a temporary or even a permanent inhibitory influence; the tremor increases, however, occasionally during such movements in the limb which is not manipulated,—*i.e.*, it is transferred to other muscles. I have even observed that the mere attempt to touch the tremulous extremity or a sudden approach to it suffices to inhibit the tremor for the time being. The same result may occasionally be secured by fixing the patient's attention upon a certain point or object. This influence is, however, only momentary, and is of no practical interest.

The tremor is always exaggerated by *mental excitement*. This is evident from the fact that the tremor becomes more prominent in the presence of others than when the patient is alone. A conversation, or the memory of some sad event, etc., has the same effect.

The tremor may be limited to an arm or one hand, or involve both upper extremities, not rarely the arm and leg of the same side, or is found in all four extremities. In the lower extremity we find particularly an exension and flexion, adduction and abduction of the foot, but the thigh musculature is also frequently affected. On the whole, the severer grades of this disease are seen in the legs more often than in the arms. The head is not always spared, and a tremor of the lower jaw, lips, tongue, etc., is not uncommon. It is rarely seen in the laryngeal, respiratory, and abdominal muscles.

The tremor generally ceases during sleep, though there are exceptions to this rule. It generally increases at bedtime, and prevents the patient from easily falling asleep.

The *chronic muscular rigidity* is an important diagnostic symptom, especially if the tremor is absent, and this forms the whole symptom-complex. The rigidity involves the musculature of the neck, the nucha, and the vertebral column especially, but may also affect the extremities and face.

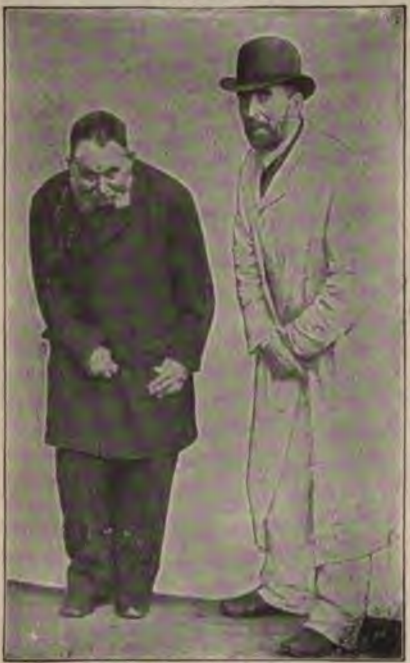
We find certain alterations of posture and position which are often the first signs of the disease, being frequently developed before passive motion is noticeably hampered. The head is generally inclined forward, more rarely to the side, and only exceptionally towards the back. The trunk is always bent forward, most of the patients having a crooked position. (Fig. 282.)

The arms are slightly adducted, the elbow-joint at an obtuse angle (rarely an acute or right angle), the hand over-extended, and the fingers either flexed at all joints, or flexed only at the metacarpophalangeal joints, while the middle and end phalanges are extended (interosseal position, writer's position). The legs are generally held in the ordinary position or are slightly flexed at the hip- and knee-joints, with slight adduction of the thigh.

Muscular contraction also produces a peculiar *statuesque rigidity of the face and the whole body*. This is especially marked when the tremor is absent, one being then reminded of a wax figure. The face may be perfectly mask-like. But we may see at first glance that it is not a paralysis of the muscles which produces it; the mouth is not opened; the angles of the mouth do not droop; the facial aspect is not a flaccid one, but appears as if turned to stone. Only the flashing eyes show the active mind.

Resistance to passive motion is always present in the later stages, and often before that time. It differs from spastic rigidity in that it is not increased by passive motion, but is constant and regular, and that slowly executed passive movements must overcome the same resistance as forcible motions. The head cannot be sufficiently rotated, flexed, and extended,—sometimes it appears completely fixed and cannot be brought out of its position. In attempts at abducting the arm, the pectoralis major and latissimus dorsi contract, and the flexed fingers in later stages may be extended, but they soon return to a flexed position, etc.

FIG. 282.



Posture in paralysis agitans.

Impairment and retardation of the active movements is a result of the muscular rigidity. As this, however, occurs before the latter appears, it may be looked upon to a certain extent as an independent symptom. The active movements are not executed as rapidly as formerly; it takes some time for the muscles to obey the will. It occurs with simple movements, but is especially noticeable in co-ordinated muscular actions. The gait undergoes a peculiar modification. The patient commences slowly to walk, but soon increases the rapidity of his gait, and strides forward as if he were desirous of falling at every step (*propulsion*). A tendency to walk backward is not infrequently met with (*retropulsion*). This occurs more particularly when the patient inclines backward, as in attempts to reach down some object from above (from a closet-shelf, etc.); he walks backward until he stops at the wall or a table, etc. This can be artificially produced by pulling the patient backward by his coat. Lateropulsion is rare.

All these disturbances, it seems to me, are due to the fact that the individual has difficulty in *bringing quickly into contraction the muscles which had previously been in a condition of rest or of tonic rigidity*. If he could bring his body, which is inclined backward, forward in time, he would not walk backward.

Paralysis only occurs in the last stage of the disease. That it does occur I was able to convince myself in many cases. But it is not an absolute one: a certain degree of motility remains.

The *deep reflexes* are never absent, but may occasionally be increased, rarely to such a degree that a clonus is produced. A false pedal tremor, as we may call it, occasionally occurs; flex the foot dorsally, and a tremor gradually ensues in the extensors of the foot and toes. It is the tremor of paralysis agitans. This paradoxic phenomenon may be produced in other regions, also in all muscles whose points of insertion are approached nearer to each other. The muscles retain their normal volume and their normal electric excitability.

The *speech* is often altered. Articulation is not disturbed, nor is there any scanning. But the voice is peculiarly monotonous, of improper modulation, without strength, and occasionally whining. It is characteristic that some time elapses before the patient speaks, but then the words follow each other rapidly, perhaps explosively. The speech disturbance is not a constant symptom. Dysarthria, aphasia, stuttering, etc., are never present.

Hyperidrosis is rather a frequent occurrence.

The bladder is not affected, though phosphaturia is said to occur quite often. Disease of the optic nerves is never found,—the cases described by König were probably complicated cases,—nor is nystagmus or ocular paralysis. In one case I observed a paralysis of convergence, which was perhaps simulated by tonic contraction of both the abducens. Sluggish ocular movements have also been described. König describes an accommodation spasm. In opening the eyes, the frontal muscles occasionally remain in a condition of tonic contraction, so that the frontal corrugations only gradually adjust themselves (Moczutkowsky).

Sensibility is not impaired objectively, and the sphincters functionate normally. The mental faculties remain intact, as a rule. If a mental disorder develops, as it does occasionally, it should be looked upon as a complication. The disease itself naturally produces depression, a tendency to cry, etc., and there is some inertia. When this is not pronounced, a certain *bonhommie* is noticed, which is in marked contrast to the severe disease. Grawitz has noticed gastric disturbances in the prodromic and early stages.

The subjective disorders result from the muscular rigidity and the limitation of active motility. The patients are therefore especially troubled at night; the inability to change their position at will annoys them and makes them dependent upon others. *Pain* is not always present, and is generally not severe; it is described as being rheumatoid, and may be an early symptom. A feeling of *heat* is often complained of, more rarely a feeling of cold. Actual increase of temperature is said occasionally to occur in the course of the disease (Fuchs).

Development and Course.—The malady usually develops slowly; only rarely is an acute onset noticed. In acute cases the anamnesis generally tells us of a sudden impulse which has passed through the arm or through the arm and leg of one side, so that it is as if paralyzed: from this time date the difficulty in motion and the tremor. On the other hand, paralysis agitans may follow a hemiplegia and confine itself to the limbs of the formerly paralyzed side.

A tremor of the type of paralysis agitans has repeatedly been observed in tumors of the region of the cerebral peduncle (Charcot, Benedict, Blocq, Marinesco). I observed it in an encephalitis of this region.

The disorder generally commences in an arm: the tremor is the first symptom noticed by the patient; it is at first slight and remittent, gradually increases in intensity, becomes more persistent, and extends to other muscles. Soon afterwards a retardation of the motility is noticed. The arm assumes a forced position, and the muscular rigidity appears in the non-tremulous parts also. It may be a year or longer before the tremor extends to the other extremities, till finally the entire musculature of the body is drawn into the field of the disease. One leg is often affected first, then the other leg or the arm of the same side.

Modifications of the above occur. The tremor is not always the first symptom. The slowing of active motion and the muscular rigidity may be the first symptoms. When it confines itself to one arm or one side it may simulate a hemiplegic condition. The disease may develop further without any tremor being noticed,—a sort of paralysis agitans without the agitation. It is in these cases especially that we notice an exception to rules laid down for the tremor. It is absent during rest, but comes on during movements,—for example, in extending the hand and finger, although perhaps only slightly.

The course is always very *chronic*. It may be fifteen to twenty years, or longer, before the patient becomes bedridden. The tremor may disappear more and more after the contracture has advanced *ad maximum*. The patient finally becomes completely helpless, and it is difficult to keep him clean. In a neglected case of my observation, the fingers were so tightly closed that the nails had fan-like continuations as long as the fingers.

In its later course, *apoplectic attacks* may occur, which, however, do not belong to the clinical picture. After apoplectic attacks which produced hemiplegia a cessation of the tremor was observed in some cases in the hemiplegic members (Parkinson, Westphal), though it generally reappears.

The prognosis is favorable as to life. Recovery is, however, not to be expected.

Differential Diagnosis.—It can hardly be confused with multiple sclerosis: the onset late in life, the character of the tremor and the contracture, the absence of nystagmus, of disease of the optic nerve, and of bladder disturbances, etc., should prevent any doubt as to the diagnosis. If the disease commences with weakness and stiffness, and the tremor is absent, the typical position of the extremities, head, and trunk should furnish a basis for the diagnosis. A slowly developing stiffness and heaviness in the limbs of one side in an aged person is generally paralysis agitans; and it is exactly this form which is so often wrongly diagnosed. The tremor of paralytic dementia does not present the typical rhythmic oscillations, continuing when the patient is at rest; the habitual posture is also absent, while the psychic anomalies, speech disturbance, and paralytic phenomena are characteristic.

Senile tremor is very similar to that of paralysis agitans; here, however, the head is especially involved, and the trembling is increased by active motion or occurs only during active motion. In addition, the other symptoms of paralysis agitans are absent.

Charcot believed that this tremor was not an attribute of old age, but occurred earlier and was identical with hereditary tremor,—a view with which I cannot agree.

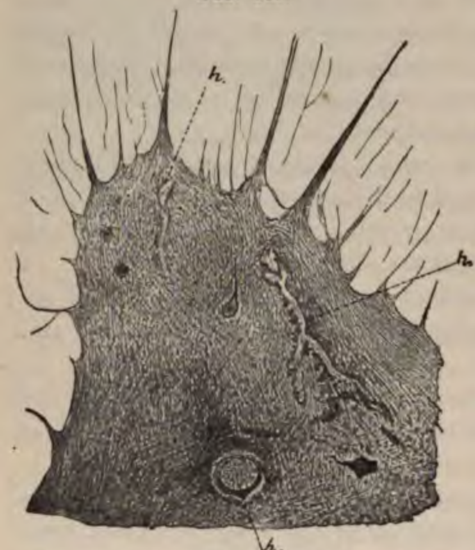
I observed in one case a combination of paralysis agitans with senile dementia and with symptoms which were produced by a focal disease of the cerebrum.

There is a form of spastic paralysis which occurs in old age (paraplegia senilis, page 209). Senile arteriosclerosis of the brain and spinal cord may in this manner produce a clinical picture which is very similar to paralysis agitans. In cases of this nature, which I treated, the posture, especially, reminded me of shaking palsy; true paralytic symptoms were, however, present, as dysphagia, dysarthria, bladder disturbance, etc. It is difficult to say just how sharply these conditions may be separated from paralysis agitans. An hysteric tremor has larger oscillations, is more dependent upon psychic occurrences, and in such cases other signs of hysteria are never absent.

It is difficult to judge those cases in which the symptoms follow a trauma. We know that paralysis agitans may be produced by injuries. On the other hand, there is *a form of traumatic neurosis* which presents the tremor and posture of paralysis agitans, but in which other symptoms are present which do not belong to the clinical picture of this disease.

Pathologic Anatomy.—We do not know anything definite concerning the anatomic basis of this disease. Most results have been negative. Recent observations (Ketcher, Borgherini, Koller, Dana, Redlich) describe alterations in the spinal cord, in the posterior and lateral tracts, and also in the gray matter, namely, sclerotic vascular processes and a perivascular sclerosis (Fig. 283) which involve, particularly, the smaller *vessels*, the *glia* (and connective tissue), and are identical with the well-known *senile processes* in the nervous system. Whether it is correct to regard paralysis agitans as an *exaggerated senile degeneration* appears to me to be doubtful. Gauthier regards it as a primary muscular disease.

FIG. 283.



Sclerotic processes of the blood-vessels and perivascular sclerosis of the gray matter of the spinal cord in paralysis agitans.

Treatment.—The physician may do *much harm* and *little good.*

In the first place everything which may excite the patient should be avoided. He should live isolated, if possible, in the region to which he is accustomed, where excitement can be avoided, and where those around him understand his trouble. These patients should not be sent to health-resorts; a sojourn in the country or woods may, however, quiet the patient.

All forced therapeutic measures should be avoided, also forced cold-water cures. Rubbing down with warm water for a short time or warm baths may do some good. Massage and electricity are of no benefit. *Electric baths*, especially the bipolar faradic, produced a certain improvement in two cases observed by me. Nerve-stretching and suspension are also useless.

All purgatives, diaphoretics, and antipyretics should be avoided. I harmed one patient with salicylate preparations, another with Dover's powder. From that time hyperidrosis began, which lasted for a long time and troubled the patient a great deal.

Tincture of veratrum viride, three to four drops, several times daily, lessened the tremor in several cases. This drug with tincture of gelsemium I can recommend. Erb praises arsenic. He gives Fowler's solution, tinctura nucis vomicæ, and aqua fœniculi in equal parts, and directs that from six to twelve drops be taken as a dose. In severe cases morphine must be given to produce sleep.

Valuable palliative remedies which produce subsidence of the tremor for some hours are hyoscin (scopolamin hydrobromate), which Erb recommended, and sulphate of duboisin which was later used by Mendel in this disease. Both drugs are severe poisons, and must be given in doses which silence the tremor without evoking any phenomena of intoxication (vertigo, heaviness of the head, visual disturbance, dryness of the throat, etc.). Hyoscin should be given in doses of two to four decimilligrams, once or twice daily, and duboisin in about the same dosage (according to Erb, a little more). They are best given hypodermically, but are also efficacious by the mouth. Erb praises hyoscin highly, but in our experience it is useful in only a few cases, and then only by continuous use, great caution being necessary to prevent toxic effects. I have also seen some good done by duboisin,—*i.e.*, a palliative effect. In one case its use caused a rapid decrease of strength and weight. Injections of arsenic are hardly of more value than its internal administration. Podack has lately recommended *sparteine.*

In the later stages the first essential is to enable the patient to secure a comfortable position and to make arrangements enabling him to change it easily. The bed-covering should be light; nourishment, strengthening and non-irritating; and alcohol allowed only in small quantities. Most patients demand some movement in the open, but excesses must be avoided.

Charcot called attention to the fact that these patients feel better when riding in carriages or on the railroad. This observation, which is not true of all cases, has led to the invention of vibratory chairs and similar mechanical appliances, whose use is, however, of little benefit.

SECTION V.

DISEASES OF THE SYMPATHETIC NERVOUS SYSTEM. ANGIONEUROSES AND TROPHONEUROSES.

ANATOMY AND PHYSIOLOGY.

THE sympathetic nervous system—the ganglion nerves, according to Kölliker—consists (1) of a chain of ganglia lying alongside the vertebral column, which are bound together by longitudinal commissural fibres into a tract extending from the head to the coccyx. (2) Of the rami communicantes,—*i.e.*, of fibres which for the most part come from the spinal cord, leave it with the spinal roots, and pass from these or from the cerebrospinal nerves formed by it into the ganglia of the chain first mentioned. The rami communicantes also contain fibres which come from the sympathetic, and pass over into the tract of the cerebrospinal nerves and accompany these towards the periphery. (3) Of peripheral branches which leave the ganglion chain at all heights, passing to the blood-vessels, abdominal organs, glands, etc. These peripheral branches anastomose with branches of the cerebrospinal nerves to form the sympathetic plexus, in which, again, peripheral ganglia are found at various places.

We divide the sympathetic nervous system into a cephalic, cervical, mammary, abdominal, and a pelvic part. The sympathetic system sends branches to all parts of the body where unstriated muscular tissue is present, especially to the muscles of the blood-vessels, stomach, intestines, bronchi, lungs, urethra, bladder, and uterus; it also innervates the dilator muscle of the pupil, the unstriated muscle of the lid, and the erectores pilorum, and sends fibres to the sweat and salivary glands. Medullated fibres from the rami communicantes may be found in the sympathetic. The cells of the sympathetic ganglia are generally multipolar, and possess an axis-cylinder process and numerous dendrites.

The fibres coming from the spinal cord—that is, most of the rami communicantes—enter the sympathetic ganglia as precellular fibres (Kölliker) to surround the cells of these ganglia with their end-brushes. Here ends the neuron of the first order. The ganglion cells send out postcellular fibres which pass to the periphery and branch in the unstriated muscular tissue, etc. They form with the original cells the neuron of the second order.

Some of the precellular fibres run directly to the peripheral ganglia of the sympathetic reticuli (Langley). This is also true of the *splanchnici.*

Physiology teaches that motor, vasomotor, and secretory (and trophic?) impulses pass from the centres of the spinal cord and oblongata by means of the rami communicantes to reach the cells of the sympathetic ganglia. From here the impulses pass to the second neuron.

The symptoms of sympathetic lesions have been particularly studied upon the cervical sympathetic, partly by experiments on animals (Cl. Bernard), and upon those executed (R. Wagner, H. Müller, G. Fischer). Injuries of this region have also served to enlighten us.

Section of the sympathetic produces, according to the noted experiments of Cl. Bernard, dilatation of the blood-vessels of the corresponding side of the head with increased temperature of the skin, contraction of the pupil and palpebral fissure of the same side, occasionally also retraction of the eyeball (enophthalmos). Durdufi claims that section of the sympathetic of the neck produces in young animals hypertrophy of the ear of the same side. Stimulation of the sympathetic produces contraction of the arterioles and decreased temperature, dilatation of the pupils and palpebral fissure, protrusion of the eyeball, also sweating of the same side of the head. The parotid glands have been stimulated and the heart's action increased by stimulation of the cervical sympathetic.

Similar disturbances have been observed in human beings following injuries, cervical operations, or lesions of the nerves from tumor-compression. Inflammatory alterations of the apices of the lungs or of the pleura are said to involve the sympathetic on some occasions. Disease of the sympathetic has also been observed in diabetes.

As signs of *paralysis* of this nerve we find the following symptoms. (1) Contraction of the pupil of the same side, with a sluggish reaction to light. (2) Contraction of the palpebral fissure of the same side. (3) Retraction of the eyeball. This symptom is not so constant as the others, and only develops gradually. It is supposed to be due to an atrophy of the fat of the orbit and to paralysis of the so-called orbital muscle, a striated muscle of the orbit but little developed in man. (4) Dilatation of the blood-vessels on the same side of the face and head. This phenomenon is not only often absent, but a contraction has also been observed. Nicati's view, that this forms a second stage and that it is preceded by dilatation, has been rejected, although physiologists have favored the same idea. It may be assumed that contraction of the blood-vessels only occurs in lesions which cause only a partial break in continuity, so that irritative symptoms are combined with paralytic ones. (5) Anidrosis of the same side of the face,—an inconstant symptom. Hyperidrosis has also been occasionally found under similar conditions. (6) Rarely emaciation of the same side of the face (Seeligmüller, Moebius).

Alterations of the cardiac functions have not been observed under such conditions. Perhaps the sympathetic of the opposite side serves to regulate the heart's action.

Moebius has called attention to the fact that pupillary dilatation may also be absent in sensory irritation upon the side of the paralysis.

Our knowledge of the symptoms of *sympathetic irritation* in men is still more uncertain, although it should be remembered that the diseases causing it always serve at the same time to produce the conditions necessary for paralysis of the sympathetic fibres. The paralytic symptoms generally follow the irritation or occur at the same time. Seeligmüller has seen a dilatation of the pupils with flattening of the brow. The dilated pupil is also said to react sluggishly (F. Pick). It must be remembered that diseases of the spinal cord (ciliospinal centre) may produce some of the phenomena described, that almost all of these symptoms may come from the cerebral cortex, and that they may also occur in the functional neuroses. It should especially be remembered that some of the phenomena, particularly pupillary dilatation, may be due to reflex causes.

We also know little that is reliable concerning diseases of the thoracic and abdominal sympathetic. We know that it contains the vasomotor fibres for the blood-vessels of the abdominal organs, that intestinal movements, evacuation of the bladder, movements of the bladder, etc., are under its influence, and that it contains motor and inhibitory fibres for the musculature of the abdominal organs.

Diseases of the sympathetic have repeatedly been observed in exophthalmic goitre.

In a very obscure case of abscess-formation alongside the dorsal cervical vertebræ, with atrophy of the sympathetic, a unilateral edema was resent during life, extending over the whole side of the body.

ANGIONEUROSES AND TROPHONEUROSES.

In some diseases, the *vasomotor* symptoms are the chief phenomena, or the only ones, without there being any known basis or origin for the malady. We call these diseases the *angioneuroses* or *trophoneuroses*.

Although many of the symptoms are transmitted through the sympathetic system, it is not said that the sympathetic forms the starting-point for the disease. To these vasomotor symptoms belong sudden attacks of flushing or pallor, with sensations of heat, rapid pulsation, and often hyperidrosis. The affected individuals complain of palpitation of the heart, rush of blood to the head, ringing in the ears, a mist before the eyes, fear, etc. Consciousness remains intact.

This symptom-complex, which, in its chief symptoms, indicates a fleeting *paralysis of the vasomotors*, occurs generally with *hysteria and neurasthenia*, though it may reach a certain independence and occur in otherwise healthy persons. Masturbation is at times a cause; sexual excesses of other kinds may also produce the disease. It may be a phenomenon of the climacteric. It should be treated by hydrotherapeutic measures and by galvanization of the sympathetic.

ACUTE CIRCUMSCRIBED EDEMA OF THE SKIN (QUINCKE), ANGIONEUROTIC EDEMA.

This disease generally attacks youthful individuals, males more often than females. It is characterized by paroxysmal attacks of *circumscribed edematous swelling* of the skin and subcutaneous tissue, occasionally also of the mucous membranes. Round intumescences, of a diameter of from two to ten centimetres, appear, which are mapped out dictinctly from the surrounding parts, and have a pale color, or may be of a deeper red than the neighboring region. Itching and pain are absent. These swellings develop simultaneously in different parts of the body and disappear after a short time, at the most within some hours. They may, however, often reappear.

The *tongue, lips, larynx, pharynx, conjunctiva*, and, as is thought, also the *stomach* and *intestines*, may be the seat of the edema. An uncommon accessory symptom is articular effusion. In a case of this kind which I saw, the suddenly appearing articular swelling simulated an attack of gout.

The general state is not disturbed, as a rule. The swellings of the mucous membranes may, however, produce much discomfort. *Gastro-intestinal symptoms*, as nausea and periodical vomiting, may thus occur, supposed to be due to an internal urticaria. *Edema of the glottis* occurs rarely, but it caused death in one case. In another case (Higier) the edema of the velum palatinum simulated attacks of pseudocroup. *Hemorrhages* from the mucous membranes occur very rarely. Joseph observed this disease in combination with paroxysmal *hemoglobinuria*. Albuminuria may also occur. In one of my cases this caused a diagnosis of kidney disease to be made.

The disease attacks almost exclusively nervous individuals, and heredity therefore plays a prominent part in the etiology; it has been observed in combination with *hysteria, neurasthenia, exophthalmic goitre, urticaria*. The disorder is very similar to urticaria. Alcoholism also seems able to induce this condition.

Exposure to cold, psychic emotion, and trauma are *exciting causes*. A sojourn on the sea seems also able to produce the trouble.

Many hypotheses have been advanced to explain the occurrence of circumscribed edema. A local *venous spasm* has been blamed. Some authors believe that the nerves have a direct influence upon the capillary cells and are able to excite these to lymph-secretion.

Acute circumscribed edema is a stubborn disorder; at least the disease has a tendency to recur constantly, and thus may last many years.

Treatment.—A general constitutional treatment is advisable. The nervous system must be strengthened. Hydrotherapeutic measures should be instituted. Digestion should be regulated. Laxatives may do good. Atropine and quinine have been used; the latter is said to have produced complete recovery in one case. In one of my cases, the use of quinine caused the symptoms to disappear rapidly. Psychotherapeutic treatment is also in place.

INTERMITTENT ARTICULAR HYDROPS.

This is a very rare disease, first described by Moore, the chief symptom of which is an articular swelling due to effusion of fluid into the joint. In most cases the *knee-joint* is involved on one side, rarely on both; the swelling may, however, involve the other joints, even the spinal column and maxillary articulations (Féré). The articular swelling appears periodically, at regular intervals, every few days or weeks; the patient being often able to tell in advance the day on which the effusion will occur. The skin over the joint is not reddened, does not feel hot; the temperature is normal; pain is not always present. A mild fever occurred in a few cases. The disease lasts, as a rule, three to eight days, the effusion then becomes absorbed and a normal condition again takes place. It may be an independent disorder, or come on in one of the general neuroses, or in combination with exophthalmic goitre, angina pectoris, etc. Trauma is also given as a cause.

In one case, flushing of the face, polyuria, and hyperidrosis alternated with the hydrops. In another case, in which the hydrops occurred in the course of exophthalmic goitre, the symptoms of the latter disappeared when the articular swelling was present. Féré observed, in an hysteric morphinomaniac, a hydrops of the knee-joint, which disappeared in a few minutes after an injection of morphine. In another case, the bilateral hydrops of the knee was combined with edema of the upper extremities. He also described a permanent hydrops. It is without doubt a *nervous* affection which has nothing to do with acute articular rheumatism, even though cocci were found in one case. Its relationship to malaria, which has been assumed in a few cases, is also doubtful.

The disease is very stubborn to treatment. In one case, the attacks occurred at intervals of nine days during eighteen years. *Electricity, salicylates, quinine, arsenic, ergotin* (hypodermically) are recommended. Laxatives do good. Hypnosis was beneficial in some cases.

VASOMOTOR NEUROSIS OF THE EXTREMITIES (NOTHNAGEL); ACROPARESTHESIA (SCHULTZE).

This disease is generally observed in females, especially at the climacteric. It is rare before the thirtieth year. Males are occasionally attacked.

As **causes** have been given exposure to cold, frequent handling of cold water or of waters of different temperatures (washerwomen), and overuse of the hands in sewing, darning, etc. Anemia, cachexia, and pregnancy may also, as it appears, be causes. Saundby blames gastric disorders. It was in some cases referred to influenza.

The symptoms are in a majority of cases entirely *subjective*. The individual complains of *paresthesia* in the hands and especially in the fingers. Formication, numbness, a feeling of their being asleep, are the sensations experienced, now this sensation, now that one being described. It is most marked in the *finger-tips*, but may, to a less degree, extend to the proximal parts of the extremity. The feet and toes are involved in only a small percentage of cases. The sensations may reach such an intensity as to be painful. The paresthesia exists incessantly, is most pronounced in the night and in the morning, before and after arising, so that fine handwork cannot be undertaken in the early hours of the day. It may confine itself to one hand or even to certain fingers. The patients seek to relieve themselves by rubbing, beating, or warming their hands. One hand or only certain fingers may be affected.

Objectively, either nothing is found, or only a slight decrease of sensibility on the finger-tips, which may be present at certain times and be absent on other occasions. A *pallor* of the fingers is rarely observed. All other functions are normal. Some of my patients, however, complained of a rush of blood to the head, cardiac palpitation, etc. In their descendants I have found upon several occasions vasomotor disturbances of another nature,—for instance, urticaria factitia.

The disease develops insidiously, rarely acutely, as it did with one of my patients.

The course is a chronic one; it lasts many years, though some cases run their course rapidly. Recovery is problematical; *spontaneous* recovery or marked improvement has occurred in some cases after many years. Life is in no way endangered.

In its early stage this disease may be confused with other diseases of the nervous system. Tabes dorsalis may commence with paresthesia of the upper extremities; the pains follow soon afterwards, however; the ataxia develops, etc. Careful examination will guard against its being confused with gliosis and other spinal disorders.

Raynaud's disease may commence with paresthesia, but local asphyxia, cyanosis, and gangrene soon follow. A similar symptom-complex, described by Rosenbach, was characterized by tubercles on the end phalanges. Some forms of tetany are ushered in with a similar paresthesia, but increased mechanical and electrical excitability of the nerves will be found present. Sternberg claims that *acromegaly* may present similar symptoms.

Hysteric paresthesia is easily excluded. Hysteric patients may also complain of paresthesia of the hands, but it is only an intercurrent, labile symptom. The influence of suggestion will also help to reveal its hysteric nature.

The paresthesia described by Berger is not identical. In this form the paresthesia occurred paroxysmally in youthful individuals, especially at the onset of movements, walking, etc., and was combined with a feeling of weakness. It consisted of a feeling of crawling or a dead sensation, of burning or pricking in a leg or both lower limbs (rarely passing to the arms), in the hips, radiating downward or ascending from the feet to the thigh. Objective symptoms were absent.

Acroparesthesia is probably due to a *condition of irritation of the vasomotor centres,* by which the arteries are contracted and the nourishment of the sensory nerve endings in the extremities diminished. It is not impossible that a mild neuritis of the nerve branches may be the cause, though the absence of pressure-points, tenderness to pressure, etc., are opposed to the view.

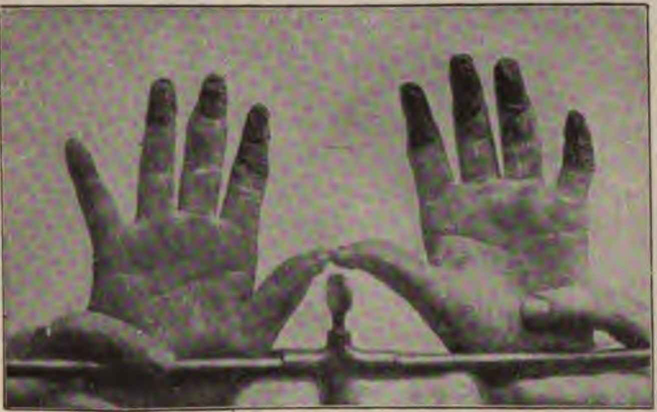
Arsenic, phosphorus, strychnine, and *iron,* galvanization of the medulla, cervical cord, and sympathetic, and local faradization have all been recommended. Quinine is also said to do good. Sinkler recommends ergotin. Overuse of the hands, excessive handling of cold water, etc., should be avoided.

SYMMETRICAL GANGRENE (SYMMETRICAL LOCAL ASPHYXIA; RAYNAUD'S DISEASE).

This is an affection which occurs *independently,* or in the course of other nervous diseases, as in *hysteria, tabes dorsalis, syringomyelia, tumors of the spinal cord and the spinal roots, epilepsy, exophthalmic goitre,* etc. A neuropathic diathesis seems to be always present. *Anemia* and *conditions of exhaustion* increase the predisposition; and, more so, a congenitally small aorta. The disease attacks mostly youthful individuals of the female sex (until the thirtieth year), more rarely males and those of mature years. Children, even sucklings, may be attacked. Emotional excitement, exposure to cold, suppression of the menses, and trauma are exciting causes. It has been observed several times to follow *infectious diseases,* and has also been noticed after lues. In one case it was referred to morphine and chloral poisoning. Some authors, as M. Weiss, seek to include under Raynaud's disease only those cases in which the disease occurred independently, and to consider those analogous affections occurring in the course of spinal diseases, infectious fevers, etc., to be distinct from Raynaud's disease. This differentiation cannot, however, be made. Others seek to include diabetic gangrene under this disease; these erring too much in an opposite direction.

The disease occurs in paroxysms. An attack is ushered in by paresthesia, with a sensation of crawling, numbness, or dead feeling in the fingers, and perhaps in the toes. The fingers become cold and pale, even waxy-white, like those of a dead person (*local syncope, regional ischemia*). Needle thrusts do not produce blood. Severe pains in the whole extremity may precede the attack for days or weeks, and increase with the onset of the seizure.

FIG. 284



Symmetrical gangrene. (After Dehio.)

The local syncope may disappear without leaving any signs, or in a few minutes or hours a *regional cyanosis*, or *local asphyxia*, follows it. A *blue-red* discoloration of the skin manifests itself on *symmetrical* parts of the hands and feet, fingers, and toes, almost entirely on the *terminal phalanges*, which become deep blue, blue-black, and finally black.

The pains increase till they become unbearable. Slight hemorrhages and bullæ may occur.

The cyanosis is followed by *gangrene*, or the blue-black color changes to a red one, and the coldness decreases (regional rubor). Rubor may also occur instead of the cyanosis. Weiss seeks to refer this to a spasm of the vasodilators. A retrogression is also possible at this stage. The gangrene commences with small *black spots*, or with vesicles filled with bloody serum, which burst and leave a superficial *black crust*, which gradually exfoliates, or an ulcer which slowly heals over. The gangrene may also extend downward so that the whole or a part of the phalanx mummifies (Fig. 284). The dead tissue is limited by a line of demarcation, and is thrown off in a few months, while the stump gradually heals. Suppuration is always slight.

The whole process occurs without any fever. The severe pains may influence the general constitution through the insomnia produced by them. *Psychic* disturbances may belong to the prodromes, especially deep depression. Gastric disorders are also not uncommon at this stage.

During the attack the sensibility in the affected parts is decreased, either for all sensory qualities or for only a few (particularly for painful and thermic stimulation); a slowness of sensory conduction has also been observed. Movements are also hampered, and are executed stiffly and slowly. Several times an atrophy of the small muscles of the hand, hypertrophy of the epidermis, and alterations of the nails occurred. *Trophic* disturbances of the joints were observed more rarely: an acute synovitis which extended over many joints and was combined with swelling of the soft parts. The temperature of the skin may in the stage of local asphyxia sink far below that of the surrounding parts.

Ocular disturbances, impairment of hearing, tinnitus aurium, albuminuria, and hæmaturia may all occur. M. Weiss has observed conditions of collape, which he refers to spasm of the coronary arteries. Signs of *sympathetic paralysis*, especially the oculopupillary symptoms, have also been noticed. Aphasia is a rare symptom.

The gangrene, as a rule, affects the end phalanges of certain fingers and toes in a symmetrical manner; more rarely all of them. An asymmetrical extension or a unilateral development of the gangrene is rare. It is not unusual to find it extend to the middle and basal phalanges. It may extend to other parts of the body,—for instance, to the nasal tip, ears, nates, thigh, etc.

An attack lasts from two to four months. This disease may then cease, which is the general rule; or more attacks rapidly follow one another, or intermissions of years may occur. Mild vasomotor disturbances are then present in the intervals. Raynaud's disease may combine with scleroderma. *Panarities* developed in the intervals between attacks in several cases.

We know little concerning the nature of the disease. It is probably a vascular spasm of the arteries and veins. Neuritic alterations have been discovered in the nerves of the fingers (Pitres and Vaillard) which are probably secondary. Dehio observed an *endarteritis* and an *endophlebitis* of the small vessels. That Raynaud's disease may occur in the course of tabes dorsalis and syringomyelia has been definitely established.

We may perhaps assume that various processes (organic, infectious, and toxic conditions) localized at definite places in the spinal cord (posterior and lateral gray matter, for instance) may cause this symptom-complex, and that in typical cases in which the disease shows an independent course they are slight, reparable injuries. It is doubtful whether it can also result from neuritic processes (Pitres, Vaillard, Hocenegg, Lancereaux).

We might also imagine a disease of the sympathetic ganglia, in that (as in erythromelalgia) a spinal-cord disease may cause the loss of inhibitory and regulating influences, so that the sympathetic ganglia exert an uncontrolled action.

Ehlers has lately claimed that this and allied trophoneuroses were due to ergot poisoning.

Diagnosis.—Local syncope does not justify a diagnosis of symmetrical gangrene, as the so-called "dead-finger" may occur without any other symptom of this disease manifesting itself. In some individuals, also, the skin of the hands and feet may become markedly cyanotic under the influence of cold, and movements of the limbs in this condition may be hampered for a certain time without gangrene occurring.

The youthfulness of the patient, the symmetry of the lesion, the sensory, motor, trophic, and other accessory symptoms, and the absence of diseases of the cardiac and vascular system make the disease sufficiently characteristic. It can easily be determined that diabetes and nephritis are not causes. The question whether symmetrical gangrene is a primary disease or whether it belongs to the symptom-complex of a spinal affection, especially gliosis, cannot always be easily answered.

In lepra mutilans the gangrene immediately involves several fingers, which fall off without pain; the specific alterations of leprosy also confirm the diagnosis.

The prognosis is, in general, favorable. The disease does not produce death. Remissions may occur. Septic infection may cause death.

Treatment.—The object of treatment should be to improve the general condition, to have the patient avoid all excitement, and to strengthen the nervous system. In the attack, bodily and mental quiet are demanded. If the pains are very severe, they must be combated with narcotics (chloral or morphine). Hypodermic injections may easily excite local grangrene, and therefore must be given very carefully. The affected parts should not be rubbed, but should be wrapped carefully in warm flannel. *Mild massage, dry warmth, lukewarm hand-* and *foot-baths*, and *Priessnitz's apparatus* are all recommended. Amyl nitrite has produced no result with me. Galvanic treatment of the spinal cord and sympathetic has been advised.

ERYTHROMELALGIA.

The condition which Weir Mitchell has characterized as erythromelalgia, and which was more completely described by Lannois, begins with *burning pains and reddening of the skin on the distal part of the feet*, more rarely on the hands, or on all four extremities.

The pains may come on suddenly, or a general discomfort or fever may precede them. They occasionally follow some over-exertion. They have their seat in the feet and toes, especially on the ball of the great toe, as well as on the heel, in the hands and fingers, and may pass to the proximal parts of the limbs. Soon afterwards occurs the reddening, which is most marked on the end phalanges of the toes and fingers. The pulp is especially reddened and also swollen. The patient complains of a feeling of heat; the skin-temperature is objectively increased; the blood-vessels pulsate distinctly; and local hyperidrosis or small nodosities are observed. The sensibility is generally not markedly impaired, though hyperesthesia, as well as a slight hypesthesia, may occur.

Redness, swelling, and pain are symptoms of variable intensity. At first the pain occurs only for a short time, especially after over-exertion of the foot; later it may be stationary or remit. It is rarely mild, but generally severe, and may increase "to real torture."

A recumbent position and cold may make it less severe, while it is increased by walking, standing, and warmth. The swelling especially is increased by walking. In standing, the feet become redder and redder, the vessels pulsate heavily, and the skin of the extremities may take on a dusky purple color; the veins appear prominently. If the symptoms are severe, the patient avoids all contact with the floor.

Accessory symptoms are generally present. Most cases complain of *headache*, *vertigo*, *palpitation*, and *attacks of syncope*. Many observations tend to show that erythromelalgia may occur in the course of a central disease of the nervous system, as, for instance, hemiplegia. In some cases, an *atrophic* condition of the muscles of the extremities occurs. A combination of muscular dystrophy with the symptoms of cerebral tumor has also been noticed (Eulenburg). More and more does it appear (Lewin and Benda) that this is not an independent disease but merely a symptom, which is due to some organic or functional disease of the central nervous system and of the peripheral nerves (neuralgia, neuritis). The disturbance rarely develops in other parts of the body. Males are the most often attacked. As causes, we may mention exposure to cold, a neuropathic diathesis, and lues. It is a very stubborn disease, and extends with remissions and exacerbations over many years. It is worse in summer.

As to the basal cause, we know nothing definite. (1) Some believe it to be an angioneurosis, due to disease of the vasomotor nervous system. (2) Others regard it as being due to disease of the posterior and lateral spinal gray matter. (3) Still others group it with the preceding diseases described as acroneuroses or acrotrophoneuroses (Hutchinson, Lancereaux). (4) Some regard it as a plantar neuritis.

Faradic currents, cold, and the use of antipyretics for the pain have been recommended.

Dehio observed the symptoms of erythromelalgia disappear after extirpation of the ulnar nerve, and drew the conclusion that it was due to an irritation of the vasodilatory centres.

There are many conditions which are allied to erythromelalgia, and yet cannot be identified with it. Among these is included Pick's case, which he styled erythromelia, of a partly circumscribed, partly diffuse reddening of the skin, which followed a marked venous dilatation, and which was found on the extensor side of the extremity.

Johannssen's case with symptoms of erythromelalgia and Raynaud's disease, and Potain's case with symmetrical gangrene on one side and erythromelalgia on the other, etc., are examples of similar disturbances. I treated a mental case in which there developed a light red discoloration of the skin of the left hand, which remained persistently without any other symptom coming on. I have also observed individuals who complained of reddening and cyanosis of the hands, which remained persistently, and which increased paroxysmally, especially in the cold, to such a degree as to become unbearable. It appears to me that different transitional and mixed forms may occur in these diseases, and that therefore a satisfactory classification presents more difficulties than in any other class of diseases.

SCLERODERMA.

This disease, which occurs at all ages, especially, however, in adult women (seventy-five per cent. of all cases, according to Kaposi), is characterized by an extraordinary induration, shrivelling, and atrophy of the subcutaneous tissue, from which the skin becomes tense, feels brawny and stiff, and becomes adherent to the underlying tissue. Disappearance of pigmentation and abnormal pigmentation are frequent accessory symptoms, also bullæ, cyanosis, marbling of the skin, and the formation of scales.

The process may be circumscribed or diffuse. The spots, at first isolated or disseminated, run together later on, so that the disease extends over a large area of the skin.

It seems to favor in its attacks the face, neck, and upper extremities.

The upper trunk is also rather often involved. It is generally bilateral. Hutchinson and Kaposi have observed it occur unilaterally. On rare occasions it follows the course of the peripheral nerves, or confines itself to the innervation area of a spinal segment. Kaposi differentiates two stages, the *stadium elevatum* and the *stadium atrophicum*. The first stage presents an edematous and hypertrophic skin, and generally escapes observation.

In the second stage we find a smooth, thin, tense, cold skin, with prominent veins, abnormal pigmentation, or absence of the same, a hard subcutaneous tissue, etc. The atrophy may extend to the deeper parts and cause knob-like hypertrophies on the bones as well as acute and chronic articular diseases.

If the process involves the hands, we may find stiffening and crippling of the fingers (*sclerodactyly*), with a thinning of the bones—a sort of acromicria—and an induration, shrivelling, and atrophy of the muscles. Circular constrictions and mutilations (spontaneous amputation) of the fingers are more rare (annular sclerodactyly of Düring). Ulcerations may occur in the later stages.

The face has a characteristic appearance, due to the smoothness and stiffness of the skin, the shortening of the lips and nasal alæ, and to the hinderance to mimic actions.

Pain, paresthesia, itching, and a feeling of tenseness are the subjective symptoms.

The sensibility is not, as a rule, disturbed. Muscular atrophy may be combined with the scleroderma and may even appear at distant places (Westphal). Paralytic symptoms are rare. The patient is often depressed. A general marasmus occurs in severe cases.

Scleroderma may be combined with progressive hemiatrophy of the face (Hallopeau, Grasset, Eulenburg, Pelizaeus), the two diseases appearing to be closely allied. It may also be seen in combination with Raynaud's disease, erythromelalgia, Addison's disease, etc.

Almost all recent authorities (Schwimmer, Eulenburg, Kaposi, etc.) consider it a trophoneurosis. Others regard it as a skin disease produced by local infection. The anatomic investigations which have been made have not revealed anything definite. We know little concerning the etiology. The **prognosis** is an earnest one. Improvement, even recovery, may occur, but in advanced cases can hardly be expected. It generally *progresses* gradually, and the patient dies from marasmus or from some complication.

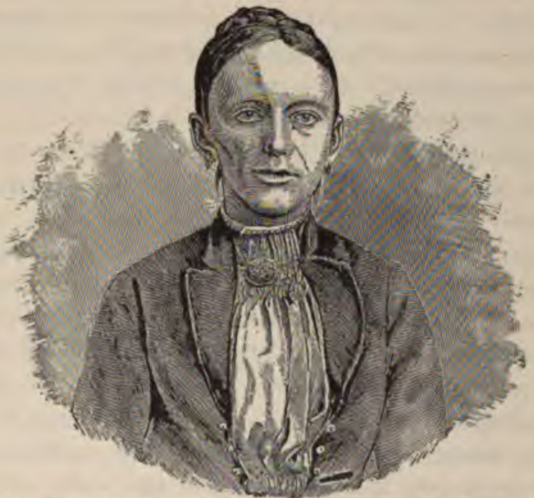
The treatment consists in massage, local and central galvanic currents, local inunctions (naphthol salicylate ointment), salol internally, and tonics.

PROGRESSIVE FACIAL HEMIATROPHY.

This disease was first described by Romberg. It is rather rare, and occurs almost exclusively in young persons from ten to twenty years of age, being uncommon after the thirtieth year. A cause cannot always be detected. Injuries to the face or cranium have often preceded the atrophy. It has several times followed an infectious disease. A facial neuralgia preceded it many times; neuralgic pains are, however, a common symptom of this disease. In two cases which came under my observation a severe neuropathic taint was found.

The hemiatrophy forms gradually and rarely attacks the entire half of the face at once. It generally commences at definite points, especially in the orbital region and lower jaw. The skin thins out at these places, and loses its color, or becomes brown, yellow, or blue. A local infiltration seems to precede the atrophy. The areolar tissue disappears also; the skin forms in folds and lies close to the bones, which also atrophy. The atrophy affects the skin, subcutaneous tissue, and bones, occasionally also the muscles, though the muscular atrophy is not a degenerative one,—*i.e.*, not combined with qualitative alterations of electric excitability. Neither does it cause any pronounced impairment of function. The result of the process is a decrease in size of one half of the face. The bones appear markedly smaller, the eye is deeply sunken in, the brow is wrinkled, and the skin is of a yellowish or brownish tinge. If the disease is in an advanced stage, the difference between the two sides may be considerable (Fig. 285).

FIG. 285.



A woman with progressive facial hemiatrophy. (After Hirt.)

The tongue and larynx (Schlesinger) have been involved in a few cases.

The eyebrows fall out or become white. The hair on the head is rarely involved. The secretion of the sebaceous glands is lessened; but perspiration may be increased in quantity, and the temperature of the skin remains normal. It has been noticed in several cases that the skin of the atrophic side did not redden with that of the rest of the body. *Anesthesia* is rarely present. The disease is progressive; it may, however, sooner or later, come to a stand-still. The designation hemiatrophy is often not adequate, as the atrophy may at times extend to the other side (J. Wolff). Hemiatrophy has been noticed in combination with many other nervous diseases.

It has been repeatedly found associated with neuralgia, epilepsy, and the psychoses, several times with chorea and spasms of the muscles of the face, jaw, and tongue, and once each with tabes, syringomyelia, and multiple sclerosis. I observed it in connection with a congenital paralysis of the ocular muscles. Occasionally the pupils of the diseased side were dilated or narrowed. In a patient of mine the pupils were not only dilated but also almost reactionless to light.

In the case illustrated in Fig. 285, I observed a convulsive tic; in addition, the patient was troubled with typical hemicrania; she had also, the cicatrix of a cyst, which had emptied itself, upon her nose (dermoid cyst). The combination of hemiatrophy with scleroderma and alopecia has also been described. It is closely related to scleroderma, and a differentiation is especially difficult whenever corresponding disease foci upon other parts of the body are combined with the facial hemiatrophy (Brunner and others). I saw such a case, in which a circumscribed atrophy of the soft tissues and bones had developed in a characteristic manner upon the back over the vertebral column. A hemiatrophy extending more or less over the entire half of the body has also been observed. In the case just described I found in addition to the facial hemiatrophy not only similar foci upon the back but also a slight muscular atrophy—without weakness and without alterations of electric excitability—in the right leg.

Most authors consider this disease to be a trophoneurosis, and regard it as being an affection of the trigeminal (Virchow), or of the Gasserian ganglion (Bärwinkel), or of the sympathetic nervous system. Bitot believes it to be a primary skin disease. Moebius looks upon it as the result of a local infection or of an infection carried here from the tonsils. This hypothesis is based upon the fact that it often follows local injuries and infectious processes in its neighborhood (angina, abscess of a tooth, etc.). We cannot, however, do without the additional hypothesis that this unknown infectious agent acts only in neuropathic individuals. The sympathetic theory cannot be so easily put aside by me. Similar types of facial emaciation have been observed in lesions of the sympathetic, and the fact that this disease is occasionally accompanied by dilatation of the pupils harmonizes with this view.

Treatment is of no avail in this disease.

Cases of facial hemiatrophy (Montgomery), as also a unilateral and crossed hypertrophy of the face and other parts of the body, have been described; also a hemiatrophia cruciata (Lunz).

EXOPHTHALMIC GOITRE (BASEDOW'S DISEASE; GRAVES'S DISEASE; CARDIO-THYROID EXOPHTHALMOS).

Graves, an English physician, first described this disease as a distinct symptom-complex in 1835, and Basedow independently wrote about it in Germany in 1843.

The disease is one of middle life, though it may occur at any age from early childhood. It affects women more often than men.

Hereditary influences can be detected in most cases, although it is rarely directly inherited. Dejerine saw it in four idifferent generations. Other nervous diseases are, however, found in the family, and nervousness or hysteria is generally observed long before the exophthalmic goitre itself appears.

In such predisposed individuals, the disease may occur without any further "direct" cause.

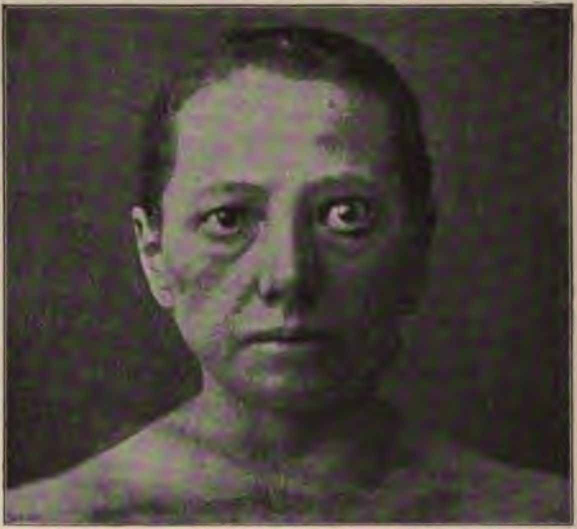
Emotional excitement or a pronounced mental shock generally, however, precedes it. Trauma, over-exertion, debilitating diseases, and difficult pregnancy may also be exciting causes. It is doubtful whether infectious diseases and syphilis can be looked upon as causes. I observed two cases develop after influenza; other factors were, however, also in action.

Symptomatology.—We speak of three cardinal symptoms,—(1) *tachycardia*, (2) *struma*, (3) *exophthalmos*. These by no means, however, constitute all the symptoms. The *tachycardia* is the most important. It occurs in all cases and in the initial stage. It may be regarded as the first symptom. The frequency of the pulse varies between one hundred and two hundred, and is about one hundred and twenty to one hundred and forty on an average. The pulse is generally small and soft, while the arteries pulsate strongly and noticeably. The frequency of the pulse is increased by mental excitement, without, however, it being entirely a result of increased mental irritability. Its constancy alone would contra-indicate this. In addition to the chronic acceleration, *attacks* of tachycardia and delirium cordis have been observed. The heart tones are loud, the apex-beat diffuse and somewhat increased.

A physical examination of the heart may reveal nothing abnormal. We may occasionally detect a systolic murmur, especially at the base of the heart; the heart in the later stages is also often dilated. Signs of a relative mitral insufficiency have been observed in some cases. Valvular defects are, however, rare, and may be regarded as complications.

Struma is rarely absent; the thyroid may also be diseased, even though it is not enlarged. The struma usually occurs after the cardiac disturbance. It is generally soft, vascular, and pulsating, though, as Moebius especially indicated, symptoms of Graves's disease may exist conjointly with those of other forms of goitre. The degree of swelling is variable, and the gland has a tendency to fluctuate in size. The superficial veins are generally dilated, the hand may occasionally detect a distinct *thrill* over the thyroid or near it, and auscultation reveals a *systolic bruit*.

FIG. 286.



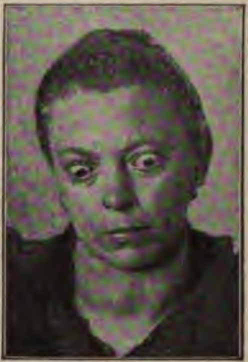
Exophthalmic goitre. Exophthalmos more pronounced on the left side.

The *exophthalmos* is, in general, *bilateral*. It may at the beginning be limited to one eye, particularly the left, or it may develop in one eye more than in the other. (Fig. 286.) It may be slight or so marked that the eyelids can no longer be completely closed; the muscular insertions become noticeable, and the conjunctiva and cornea become the seat of inflammatory processes and even of ulcers. In some cases this has made enucleation necessary.

A complete absence of exophthalmos is not so rare; when the goitre is also absent the diagnosis may present difficulties. There are, however, other symptoms which occur rather regularly, and may be classed as typical symptoms. They are the *psychic symptoms, nutritive disorders, physical weakness, tremor, skin discoloration*, and *diarrhea*.

Ocular Symptoms.—Graefe's symptom is often present. If the patient is asked to lower his eyes slowly, the upper eyelid does not follow the movement of the eyeball, or follows it but slightly, and the supracorneal part of the sclera comes into view. (Fig. 287.) There is occasionally an insufficiency of the internal recti. If the patient is asked to look at the bedclothes and then at the tip of his nose, we find that one eyeball is in a position of convergence while the other one inclines outward. This sign is of importance only when no refractive errors exist.

FIG. 287.



Exophthalmic goitre, showing Graefe's symptom. (After Bruns.)

The palpebral fissure may be abnormally wide (even with absent exophthalmos), and winking does not occur as often as in healthy persons (Stellwag's symptom). In one of my cases this phenomenon existed in only one eye. On the other hand, in some cases repeated winking has been observed. Ocular movements may be hampered by the exophthalmos; a real paralysis is rarely observed. Even ophthalmoplegia externa has, however, been noticed (Warner, Ballet). *Lacrymation* may be a symptom of this disease (Berger). A vibratory tremor of the eyeball was observed several times. Jendrassik noticed bulbar symptoms in a few cases. Occasionally the retinal arteries pulsate distinctly, though an ophthalmoscopic examination is otherwise negative, except in complicated cases. Optic atrophy has been described in two cases, but was probably a complication. The same is probably true of an optic neuritis which I noticed in one case.

Psychic Anomalies.—The mental condition in exophthalmic goitre is rarely normal. Abnormal excitability, irritability, forgetfulness, hastiness, restlessness, and confusion are almost always present. Even the facial expression and manners of the patient betray this. He looks shyly at the examiner, appears restless, his glance is wild, his answers are ambiguous and evasive, and he cannot recollect many things, etc. The degree of this disturbance varies greatly in the different stages as well as in different individuals.

Mania, melancholia, and *hallucinations* occur more rarely. They are generally atypical types of insanity.

In severe cases, and especially in those with an unfavorable course, an acute delirium may develop late in the disease. On the other hand, psychoses occur in whose course certain symptoms of Graves's disease appear. I have seen such a case, and Thoma has lately described a menstrual psychosis with periodic struma and exophthalmos. Sleep is generally disturbed and light. Several times I observed an abnormally deep sleep, accompanied by enuresis, from which the patient could not be awakened.

Nutritive disorders are always present. Emaciation may be slight or may increase to marasmus, even when the amount of food ingested is considerable, or increased in amount (Mueller). The bodily weakness of which the patients complain is partly due to this emaciation, though a real motor weakness does occur which occasionally affects the legs alone, and which may be so pronounced or temporarily so much increased that the patient collapses. A persistent paraparesis has also been observed. Hemiplegic and monoplegic conditions are very rarely present. A real *muscular atrophy* is rare. I found an almost complete gluteal atrophy in one case.

The *tremor* is an almost constant symptom, although it is not present throughout the disease. It is found in the extremities, in the trunk, or is limited to the head and upper part of the body. It is a rapid, vibratory tremor, of from eight to ten oscillations a second, the individual oscillations being regular, though slight exacerbations may occur. It is increased by motion, more so by mental excitement, though it is also noticeable during rest. In many cases, in addition to the tremor, movements similar to those of chorea were noticed.

The deep reflexes may be increased or decreased. In a few cases they were temporarily abolished. Marked pulsation of the small arteries, capillary pulse, and pulsation of the liver and spleen are occasionally observed (Gerhardt).

The *trophic, vasomotor, and secretory* disturbances are various.

A dirty discoloration of the skin is of frequent occurrence. Pigment spots, vitiligo, urticaria, and other efflorescences are also often seen. The discoloration may become as marked as in Addison's disease. In one of my cases in which the buccal mucous membrane also showed an extensive pigmentation, I diagnosed exophthalmic goitre with Addison's disease. Graying or falling out of the hair is often observed.

Local *edema* (of a nervous origin) at uncommon places is a rare occurrence. The *cardiac weakness* in severe cases finally produces a general hydrops. Swellings of a myxedematous character also occur. A swelling of the upper eyelid is a rather frequent form of the local edema. In a case examined by me, this edema of the upper lid was the first symptom noticed. Nasal hemorrhages are not so rare; internal hemorrhage is. Multiple telangiectases occurred in one case.

Among the secretory anomalies, hyperidrosis is the most important, as it is absent in but few cases. The sweats are partial (palms of the hands, head, etc.) or general; the secretion may be so excessive as to compel the patient repeatedly to change his clothes during the night. A hyperidrosis unilateralis occurs more rarely. The *decrease of electric resistance of the skin*, discovered by Vigouroux, is probably due to this pronounced dampness of the skin. Yet it does not occur in other diseases where the perspiration is abnormal. It is, at any rate, not a pathognomonic sign.

Exact observations (Moebius and myself, Kohler, and others) have revealed the following:

1. The absolute resistance-minimum is very low.

To understand this it is necessary to remember that the electric resistance of the skin decreases under the influence of the galvanic current. Upon the use of a moderate current strength (about ten elements) the resistance is decreased to a definite quantity (relative resistance-minimum); upon increasing it, one is able to decrease the resistance until finally a lower limit is reached, above which further increase of the number of elements does not lessen the resistance (absolute resistance-minimum).

2. The resistance-minimum is obtained with a relatively slight electromotor force (twenty-five elements,—thirty to thirty-five normally).
3. Slight decrease with slight electromotor force.

Polyuria is often present. Albuminuria and glycosuria have been observed. *Polydipsia* also occurs. *Amenorrhea* is rare. The digestive apparatus is generally impaired. The complaint concerning loss of appetite is heard as often as is that of bulimia. Attacks of vomiting occur in the course of the disease, and may increase to a condition of persistent vomiting. Diarrhea may become a very bothersome symptom. It consists of sudden and frequent and generally painful, watery stools, about four or five, sometimes ten or more in a day. They tend to produce exhaustion. Intestinal hemorrhages and hematemesis may also occur.

Respiratory disorders occur occasionally, especially dyspnea and a paroxysmal dry cough. Bryson emphasizes the lessened expansion of the chest in inspiration, particularly in the severe cases, though the respiratory weakness goes hand in hand with the muscular weakness (Patrick). The body temperature is generally normal; intercurrent attacks of fever may, however, occur. A feeling of heat is often complained of. Swelling of the cervical glands has often been observed (Gowers, Müller).

Complications.—We have already alluded to the complication of this disease with the psychoses and with cardiac lesions. It is most often seen with hysteria, occasionally with epilepsy, and rarely with tabes, tetany, diabetes, etc. Icterus may occur during its course. It may combine with or pass into myxedema (Kowalewski, Sollier). Gangrene occurs rarely. Koppen and Revilliod described osteomalacia and other alterations of the osseous system as occurring in Graves's disease.

Course and Prognosis.—It is a chronic disease, but may occur suddenly, especially after emotional shocks. A neurasthenic condition generally precedes the disease, the cardiac troubles appearing first and being followed by the other characteristic phenomena. Moebius believes that the thyroid disease is the primary alteration, but that it may remain latent a long time. Its further course is a protracted one. It lasts for years, with remissions and intermissions. Trousseau and, lately, F. Müller have described acute cases ending in death in one and one-half to two and one-half months. In a case described by Mackenzie, death is said to have resulted in three days. Recovery may also occur soon after the onset.

The duration of the disease, the severity of the symptoms, the general condition, the position in life of the patient, are all factors influencing the *prognosis*. Partially developed cases of a mild intensity, with slight psychic involvement or but slight impairment of the general nutrition, have a relatively good prognosis. If it is possible, in addition, for the patient to secure the conditions of life that are necessary to recovery, improvement at least can be assured. I have seen a number of such cases with favorable terminations. In one of these cases recovery lasted twelve to sixteen years; in another, eight years; in a case described by Chandle, twenty years. The prognosis is very bad, however, when the disease has lasted for years, is completely developed, and the individual symptoms are very marked. Marasmus, dilatation of the heart, irregularity in the heart's action, and valvular disease (one must be very cautious in Graves's disease in diagnosing valvular disease) also cloud the prognosis; also deep discoloration of the skin, and mental disorders, as well as an acute onset and an acute course. The tachycardia always serves as a good index in making a decision.

We are never justified, however, in regarding the prognosis as absolutely bad. Improvement and even recovery may occur in the worst cases. Death is a result of the heart disease, or may occur from general weakness, or at the height of a mental disorder. Continued vomiting and profuse diarrhea may also cause death.

Improvement is more frequent than recovery. Some cases recover with the exception of a slight *exophthalmos* or an almost imperceptible swelling of the thyroid. I have seen recovery occur in the severest cases. In a fifty-year-old man who complained of conditions of oppression, I found a dilatation of the heart with tachycardia, and learned that nineteen years before he had had Graves's disease with complete development of all cardinal symptoms. In one of the worst cases that I have ever seen, in which general hydrops came on, due to a severe cardiac weakness, recovery resulted. In another complicated case, in which disturbances of compensation likewise came on, an inunction treatment—the patient had syphilis—wrought a marked improvement. In another case the symptoms disappeared, but were replaced by writer's cramp and other nervous disorders. Pregnancy has an unfavorable influence on the disease, though in several cases improvement was observed in the course of the pregnancy.

Differential Diagnosis.—Typical cases are easily diagnosed. The exophthalmos, if we exclude retrobulbar tumors and aneurisms, is rarely seen in any other disease. Difficulty in diagnosis therefore occurs only in the abortive forms, when two of the cardinal symptoms, the struma and the exophthalmos, are missing, or one is but slightly developed. In such cases we may be compelled to make a diagnosis from the tachycardia and the minor symptoms. But as psychic defects, excessive perspiration, thirst, polyuria, vomiting, diarrhea, etc., occur also in neurasthenia and hysteria, as does nervous cardiac palpitation, we are liable to err in our diagnosis on this account. In these conditions, however, the tachycardia is not a constant but a paroxysmal symptom. Graefe's sign, the pigmentation, decrease in electric resistance, etc., may also, if searched for, be found present. The shyness, mental confusion, and weakness of the memory of Graves's disease are also more marked than in hysteric cases. A *myopic* eye should not be confused with exophthalmos. In doubtful cases the patient should be compared with photographs taken in earlier years.

The symptoms which a struma produces by pressure on the sympathetic nerve are, as a rule, unilateral. Suffocation, hoarseness, dysphagia, dilatation of the pupils, etc., are also the principal symptoms. It is, however, not rare for the symptoms of exophthalmic goitre to engraft themselves on an old struma (Moebius). Marie desires to make a distinction between the " Basedowifien goitre" and true Basedow's (Graves's) disease. I have seen cases in which I could not determine whether exophthalmic goitre or struma with symptoms of compression was present. The combination of a simple struma with nervous tachycardia may also obscure the diagnosis.

Acute iodism may cause a symptom-complex allied to Graves's disease.

Pathologic Anatomy.—*Nature of the Disease.*—Formerly degenerative alterations of the sympathetic were repeatedly observed and thought to be of moment. It has been noticed, however, that they are inconstant and not important. Examinations of the thyroid (Rénaut, Soupault, etc.) revealed cirrhotic processes, hyperplasia, and obliteration of the lymph-vessels. These alterations were also found in thyroids that macroscopically showed no changes. Brissaud claims that the goitre in Graves's disease does not differ pathologico-histologically from the ordinary goitre. An hypertrophy or an abnormal persistence of the thymus has been often observed in this disease.

Theories as to the nature and basis of this disease are numerous. The view that it is a disease of the sympathetic nervous system has much to recommend it. Many of the symptoms can be referred to disease of this system. The tachycardia, dilatation of the blood-vessels, the vasomotor and secretory disturbances, etc., could be referred to a disease of the sympathetic; it is, at any rate, unreasonable to take exception to this view because some of the symptoms are due to irritation, others to paralysis. Such a combination of the two has been seen in many nervous diseases. Abadie has lately taken up the *sympathetic* theory again, and looks upon the disease as being due to a *stimulation of the vasodilators* of the sympathetic system.

Another theory places the seat of the disease in the *medulla*. The experimental examinations of Filehne, who produced symptoms of Graves's disease by section of the restiform body in animals, speak for this theory. Durdufi and Bienfait came to similar conclusions. The fact that ophthalmoplegia and bulbar symptoms occasionally occur in the course of exophthalmic goitre also supports this theory. Attention has been called to the fact that in moments of pronounced excitement a complex of physiologic phenomena may come on,—the eyes become widely opened, the bulbi become prominent, the heart beats rapidly, etc.,—symptoms which are found constantly in Graves's disease in a more marked manner. The pons and oblongata may contain a centre, stimulation of which produces this symptom-complex (Putnam). Moebius, Brissaud, and others consider this region a place of minor resistance to the poison of exophthalmic goitre (see below).

Gauthier and Moebius have lately set up another theory, to which Joffroy, Marie, Revilliod, and others have agreed. According to this hypothesis, the disease is due to *overactivity of the thyroid gland* (hyperthyroidization or dysthyreosis). This theory is based on the assumption (the truth of which is now recognized by all but Munk) that the thyroid is an indispensable organ which produces substances necessary to the organism or which have the function of neutralizing poisonous products of metabolism. It has been experimentally shown that the ingestion of thyroid extract in excess by an animal will produce most of the symptoms of Graves's disease (Ballet, Lanz, de Enriquez). Buschan, however, claims that after taking an overdose of thyroid tablets he did not experience any departure from his natural state.

At any rate, the hypothesis which refers the disease to an abnormal increase in function of the thyroid gland is not very convincing. The further assumption must be made that the thyroid gland in nervous individuals is more easily placed in this condition and that nervous excitement is particularly liable to spur this organ to increased activity. The experimental attempts to excite the secretion of the thyroid gland by stimulation of the nerves have not been successful (Hürthle).

The opinion that toxic products play a *rôle* in Graves's disease seems to have some support from the detection of poisonous bodies in the urine of such patients (Chevalier, Boinet, and Silber). The idea that destruction of the lymph-spaces in the thyroid gland causes a flooding of the venous blood with the secretory products of the thyroid is also worthy of being noted. Some observers seem to think that an acute post-infectious thyroiditis is the origin of the disease (Reinhold). Finally, the fact (in favor of Moebius's theory) has been brought forward that Graves's disease may pass into myxedema (Sollier, Baldewin, Kowalewsky, Joffroy).

Lemcke assumes that the hypothetical poison acts first upon the musculature and produces an atony of it.

Concerning the products of the thyroid gland, see page 869.

Treatment.—The chief task of the physician is to place the patient under favorable conditions of life. If it is possible to close the source from which arises the psychic excitement affecting the patient, much will have been done. But even where this factor is absent or when it cannot be removed, *mental and physical rest* is the first essential. For this reason alone it is often advisable to remove the patient from his family and occupation and to place him in other surroundings. A sojourn in the country, in a hospital, or in a sanatorium may be very beneficial. Everything which may excite the patient—speculation, card-playing, theatre-going, etc.—must be interdicted.

The physician's personal influence may also aid considerably in quieting the patient. Mild exercise in the open air is allowable, even advisable. Long journeys, the climbing of stairs, etc., should be avoided. In severe tachycardia rest in bed is demanded. *Coffee, tea,* and *alcohol* should not be allowed the patient. The diet should be strengthening, non-stimulating, and almost entirely vegetable. Smoking and sexual pleasure should be forbidden.

In recent, not severe, cases, a mild cold-water treatment, especially cold rubbings, and warm baths may do good. Wet packs may relieve the tachycardia. Winternitz especially praises hydrotherapeutic measures. Sea-baths are contra-indicated, but a sojourn at the sea-shore may do good; also high, dry altitudes (up to one thousand metres), for example, in Aussee, Triberg, St. Blasien, Giessbach, etc. Improvement is also said to have occurred at St. Moritz. Mountain-climbing is, however, not permissible. *Electric treatment* is often productive of much good. The best method is the *stabile galvanization of the sympathetic.* Place the cathode electrode (of a diameter of two to three centimetres) externally to the large cornu of the hyoid bone, between the angle of the lower jaw and the inner border of the sternocleidomastoid; the other and larger one should be placed about the height of the fifth to the seventh cervical vertebra. Introduce a gradually increasing current of two to three milliamperes, shutting it off after two or three minutes. I and others have had good results with this method. Be careful not to use strong currents. Vigouroux recommends the following method of faradic treatment: broad anode of seven to eight centimetres in diameter on the neck, small cathode of one centimetre in diameter upon the sympathetic, for about one and a half minutes on each side, with a current just strong enough to cause contraction of the sternocleidomastoid; then upon the motor-points of the orbicularis palpebrarum muscle; next use a larger cathode upon the jugular, the thyroid, and the cardiac region for about ten or twelve minutes. This treatment should be carried out every second day for weeks or months. Static breezes in the cardiac region are also said to do good.

Many drugs have been recommended; none are trustworthy. Iron, quinine, belladonna, and potassium iodide may be tried. Potassium iodide must be used cautiously. Kocher and Trachewsky laud sodium phosphate in doses of two to ten grains a day. The *bromides* should be given for the tachycardia as well as the restlessness (fifteen to thirty grains three times a day). *Cold compresses* or an ice-bag over the heart also do good. Opium is not of any value for the diarrhea. I had better results with colombo; the bromides are also sometimes beneficial. If Moebius is right in claiming that the body seeks to remove the poison through the bowels, we are not justified in combating the diarrhea.

Atropin may be given for the hyperidrosis, but a hydropathic and electric treatment will accomplish more.

The exophthalmos, if considerable, may render necessary a pair of glasses to protect the eyes. Coughing, straining, etc., should be avoided, if possible. Compression of the eyeball is useless. Conjunctivitis, traumatic keratitis, etc., should be treated in the ordinary manner. The exophthalmos may make a *blepharorrhaphy* necessary.

A modified Mitchell rest-cure should be used against the marasmus.

I have never seen hypnotism do good in these cases. Moebius, however, reports some good results.

Treatment of the nose has done good in many cases. All hypertrophies, etc., of the nose should, therefore, be appropriately treated. Symptoms of Graves's disease have, however, also been produced by such nasal treatment (Schmidt, Semon). Improvement has also occurred after gynecologic operations.

In the last few years attempts have been made to treat exophthalmic goitre with thyroid and thyroid extracts, a treatment which is opposed to the above thyroid theory. A small number of cases have been apparently improved thereby; but in a majority of the cases it produced no results. My observations have also made me opposed to the use of this drug in these cases.

Owen, Mikulicz, and others have administered thymus gland (ten to fifteen grains) with good results.

Ballet and Enriquez used the serum of dogs whose thyroids had been extirpated, and found that it produced good results. We are hopeful of some day finding a substance which, when introduced into the body, will destroy the poison.

Surgical measures directed against the struma have been recommended for the cure of the struma and of the disease itself. Electrolytic treatment of the struma has produced a decrease in its size. In recent years many operations have been devised to make the thyroid harmless, either by direct extirpation (strumectomy) or by other methods.

Poncet's exothyropexy may be ignored, as it probably is only of ephemeral interest. Partial ablation of the struma was first performed by Tillaux and Rehn. In recent years many surgeons have reported good results. Heydenreich's statistics give a favorable result of the operation in eighty-two per cent. of the cases. Starr collected one hundred and forty operated cases, among which twenty-three were fatal, forty-five improved, three unchanged, and forty-five had an uncertain result.

The operation is dangerous to life. According to Buschan, fourteen per cent. of all cases operated upon die. The operation is still under judgment. It should be carried out only in severe cases when other methods have failed and where no cachexia or marked cardiac weakness is present.

Marie desires to limit strumectomy to cases of "goitre with secondary Graves's symptoms."

French authors (Abadie, Jaboulay, and others) recommend section of the sympathetic below the upper ganglion. Notwithstanding reported good results, I cannot speak favorably of this treatment.

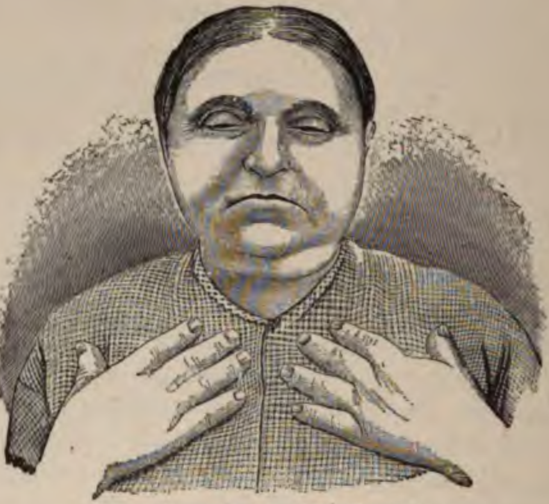
MYXEDEMA (GULL, ORD); CACHEXIE PACHYDERMIQUE (CHARCOT).

Gull and Ord in the seventies called attention to a disease which was especially characterized by a peculiar *swelling of the skin* and *subcutaneous tissue.*

This swelling occurs first and chiefly in the *face*, then in the lower extremities, and later in the upper ones.

The face appears *swollen, pale, broad,* and *full.* The swollen eyelids droop; the nose is thick and plump; the face appears apathetic and stupid (Fig. 288). The tongue becomes thickened and plump. Swellings appear on the neck and extremities. The latter are swollen out of shape, and remind one of paws. The skin is pale and cold; it feels *hard* and *elastic*, and does not pit, a true edema being nowhere evident. Perspiration ceases, the hair and nails fall out, the skin becomes dry and hard and scales off. At certain places, over the clavicles, nucha, etc., tumor-like infiltrations may occur. An infiltration of the mucous membranes and falling out of the teeth are also often encountered. The patient is clumsy and awkward; this impression is increased by the *muscular weakness* and *decreased intelligence.*

FIG. 288.



Case of myxedema. (After Charcot.)

Retardation of mentation, speech, and motility, especially an awkward wabbling gait, are characteristic signs of this disease.

Loss of hearing and amblyopia may occur. The other higher senses may also be involved. Ophthalmoscopic alterations (optic neuritis, atrophy of the optic nerve) have only rarely been observed (Wadsworth).

Albuminuria, glycosuria, synovitis of the knee-joint, etc., are rare symptoms.

The body temperature generally becomes *subnormal;* the patients also generally complain of a sensation of cold.

Of the subjective symptoms the following are the most common: *headache, forgetfulness, vertigo,* weakness, and hemorrhages.

Anesthesia often occurs; muscular atrophy, incoördination, contracture, etc., more rarely. The reflexes are generally normal; also the electric excitability of the nerves and muscles. An increase in the electric resistance of the skin has been noticed. The voice becomes hoarse, raw, and monotonous.

The *thyroid gland* is absent or very small. A swelling of it may, however, precede its disappearance. At times there is a marked tendency to hemorrhage from the nose, gums, or in the skin.

As a rule, the psychic alteration consists in simple dementia, or perhaps in irritability and hallucinations. In some cases, however, mania, melancholia, and hallucinatory paranoia, etc., have been observed (Savage).

Myxedema affects women more often than men (one hundred and seventeen females to ten males). It develops *insidiously,* and is slowly *progressive.*

The increase in breadth of the whole body, the swollen, broad, expressionless face, the monotonous, hoarse voice, the thick hands and feet, the slowness in mentation and movements, the general physical weakness, etc., are all symptoms so characteristic that a diagnosis is easy. One should always decide, however, whether myxedematous swelling or edema is present. It should not be confused with the stabile edema occurring after chronic erysipelas or with syphilitic edema.

The only results of pathologico-anatomic examinations to which any significance can be attached is—excluding the disease of the skin and subcutaneous tissue—the *disease of the thyroid gland.* Decrease or atrophy of the thyroid gland is noticed in almost all cases. Clinical and experimental observations of various kinds also indicate that the myxedema is caused by loss of the thyroid gland. This has been brilliantly proved by the modern treatment of the disease.

Kocher and Reverdin showed that a disease-complex (*cachexia strumipriva*) developed after total extirpation of the thyroid. After days, weeks, or months, during which time a tired feeling, pains and heaviness in the limbs, sensation of cold, etc., are experienced, temporary swellings of the face, hands, and feet occur.

Gradually the swelling and puffiness, which present all of the characteristics of myxedema described above, become chronic. The movements become slow, also the speech and mentation. The skin becomes cold, pale, dry, scaly; the hair falls out; the bones cease to grow; the loss of strength gradually becomes more marked; and if not treated the patient, as a rule, dies.

The disease does not manifest itself when only a part of the gland is ablated. It should not be forgotten that the symptoms of cachexia strumipriva do not always occur after total extirpation, or sometimes to only a slight degree, due probably to the fact that the so-called accessory glands assume the funtions of the thyroid. It is also known that young individuals are affected more than older ones by loss of the thyroid.

As the symptoms of cachexia strumipriva (and thyropriva) are identical with those of myxedema, there can be no doubt that the cause of this disease is *loss of function of the thyroid.*

We regard it as very probable that this gland forms a secretion which neutralizes toxic products of metabolism. The latter, if undisturbed, produce myxedema. They are especially deleterious to the central nervous system.

We know now that *sporadic cretinism* is a disease identical with myxedema, an *infantile myxedema.* Persons with this disease present the following picture: dwarf stature, idiocy, myxedematous skin, pug nose, thick and everted lips, thick tongue, drooping eyelids, short plump neck, short unformed body, and absence of hair-growth. Fatty tumors in the cervical region and umbilical hernia often occur. Walking may be impossible, speech is impossible; the development, both mental and physical, is extraordinarily slow, dentition is delayed, and the signs of pubesence never appear, etc. The absence of the thyroid is the cause of the disease.

Many of the cases of so-called "infantalism" (Bourneville, Thibierge, Brissaud) belong here.

Degeneration of the glandular tissue of the thyroid gland (athyreosis) is the primary cause of myxedema. The result of such atrophy is a hyperplasia of the connective tissue in the skin, and an accumulation of mucin in the tissues (Halliburton, Thierfelder), though it has not been proved that the swelling of the skin is due to the increased mucin in the tissues.

As exciting causes, exposure to cold, emotional excitement, traumata, difficult delivery, syphilis, and especially heredity, may be enumerated.

The prognosis is, on the whole, unfavorable, though several times spontaneous improvement, if not recovery, has occurred.

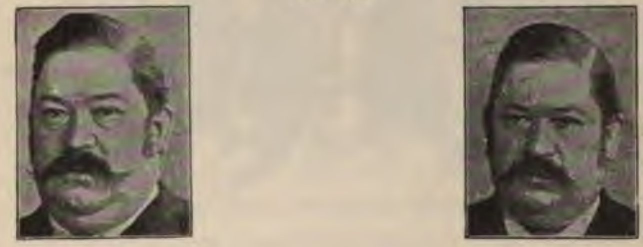
We do not yet know the therapeutic value of our modern treatment of myxedema, but the results of the last few years indicate much for the future.

Treatment.—Schiff, by his experiments, first showed that the results of thyroid extirpation do not occur if the thyroid of an animal of a similar species be implanted in the abdomen so that it continues to functionate.

Bircher, Horsley, and others built up our modern treatment of myxedema on this discovery. Originally, thyroid tissue from anthropoid apes, sheep, and calves was transplanted; later, an extract from the thyroid gland was injected subcutaneously (Murray), and next the glandular tissue or an extract from it was given internally (Howitz, Vermehren, etc.). The active substance, thyroidin, is contained in a glycerin extract and is taken from it. Baumann's discovery, that the thyroid contains an organic iodine compound, thyroiodin, which is an effective part of the gland, is very important. Neither this preparation nor others, as the thyroidin, etc., can equal in efficiency the whole organ (Gottlieb, Drechsel, Stabel, Wormser, and others).

If the gland be administered in substance, commence with a fraction of a grain and gradually increase to one or two grains. Some authors recommend much larger doses to be given twice weekly, and not daily. Powders and tablets are now on the market and can be given, as they are reliable (Ewald). Fig. 289 shows the results obtained in a short time.

FIG. 289.



A. B.

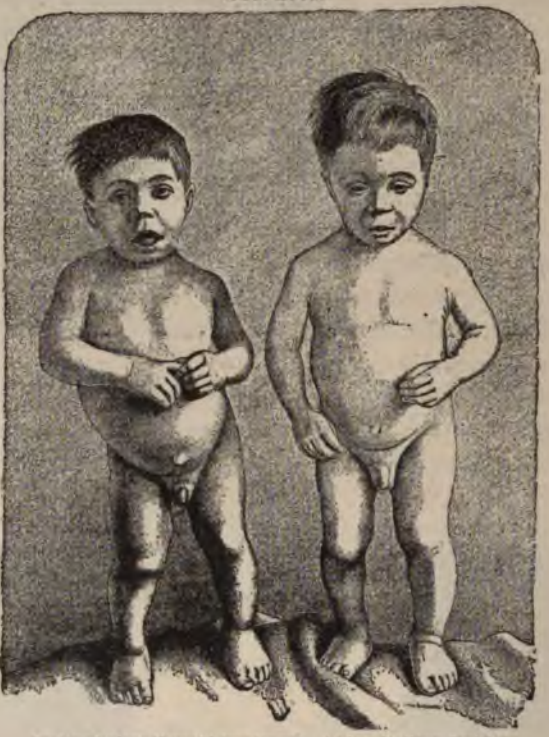
Result of thyroid feeding in myxedema: A, before treatment; B, after several weeks' use of thyroid tablets.

The patient should be placed upon a vegetable diet, and the treatment in general requires careful watching, as it may be dangerous. Though large doses of thyroid substance may be taken with impunity (Buschau, Becker), they produce, as a rule, tachycardia, dyspnea, anorexia, emaciation, etc.

Even in those affected with myxedema, unpleasant symptoms may follow the ingestion of thyroid preparations, as cardiac palpitation, tachypnea, thirst, loss of strength, albuminuria, and even uremic symptoms. There is, however, no doubt that many of these cases were due to the fact that decomposed thyroid glands were used (Lanz, Stabel).

All in all, the results of the thyroid therapy in myxedema are certain and brilliant. Relapses occur, however, when the treatment is discontinued, and require a renewal of the same. It further has been shown that a warm climate is beneficial to this class of patients.

FIG. 290*a*.



Cretins before thyroid treatment. (After Railton-Smith.)

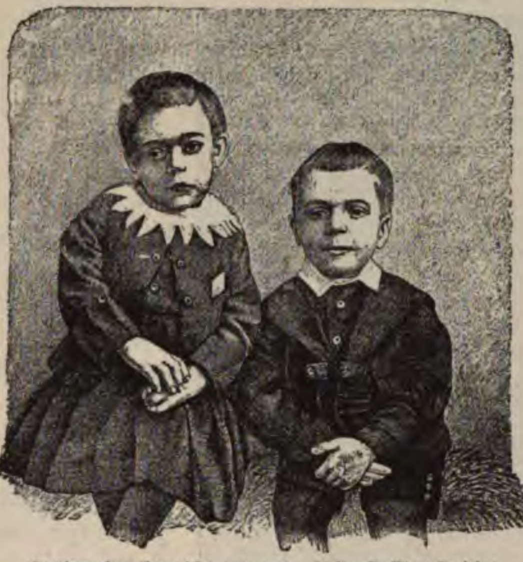
Kocher and Lichtenstern treated with success cases of cachexia strumipriva in the above manner.

Sporadic cretinism has also been successfully treated with thyroid extracts (Murray, Carmichael, Bramwell, Osler, Bourneville, and others). Figs. 290*a* and 290*b* show the remarkable result in a pair of cretins (brothers) who had been treated with thyroid tablets. Here also, however, it was not a cure, but a marked improvement which was produced.

ACROMEGALY (MARIE); PACHYAKRIA (RECKLINGHAUSEN).

This disease, first thoroughly described by Marie in 1886, may occur at any age, but is found particularly between the ages of twenty and forty. Both sexes are about equally liable to it. Traumata and emotional shocks have been given as exciting causes. It develops insidiously in most cases, commencing with drawing pains and parasthesia in the limbs and a feeling of general weakness. Cessation of the menses is sometimes an early symptom in women. The important characteristic phenomenon is *an enlargement or thickening of the distal ends of the extremities*, as well as of the nose, lips, lower jaw, etc. The enormous development of the hands and feet, and particularly of the fingers and toes, is very noticeable. The swelling involves both the bones and the soft parts, so that the hand is enlarged *in toto*, thickened, and uncouth looking, but not deformed, and the thick, plump, occasionally lengthened fingers resemble sausages. Marie makes a distinction between the long-handed and the wide-handed forms. The skin is thickened, moist, spongy, and bloated. The bones of the wrist and ankle-joint may be thickened, while the metacarpal and metatarsal bones are not particularly altered. The long bones of the extremities are also not much enlarged, so that a giant-hand is found on a slender forearm. The vertebral column, clavicles, and sternum are, on the contrary, often affected. Kyphosis of the lower cervical and upper dorsal vertebræ is very often observed, the ends of the spinous processes being also often thickened. The sternum is broadened *in toto* and thickened, the ensiform process being particularly prominent, so that the sternum presents a double hump (Sternberg). The thickening of the manubrium may cause a muffled tone on percussion, which Erb refers to the thymus.

FIG. 290*b*.

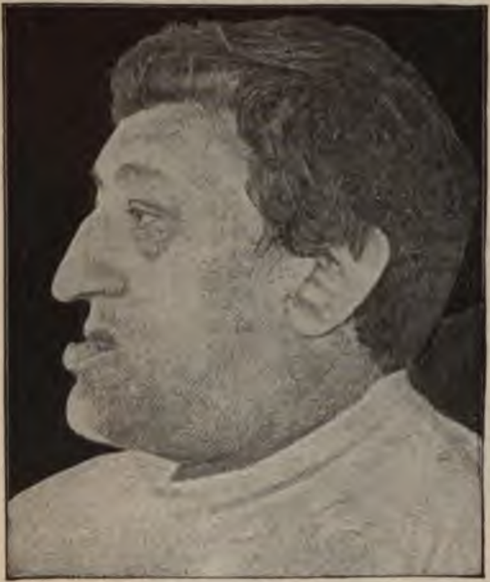


Cretins after thyroid treatment. (After Railton-Smith.)

The ends of the clavicles are thickened and elevated; the ribs are massive, as are also the patellæ.

On the head (Fig. 291) the facial bones are particularly altered. The external occipital protuberance may become very prominent (Schultze), and the cranial circumference may also be considerably increased. The projection of the chin and the occasional encircling of the upper jaw by the alveolar processes of the lower jaw is particularly characteristic (prognathism). The zygomatic arch may also protrude markedly, and the soft structures of the face become hypertrophied. The nose is thickened, turned up, and flabby, the lips are heavy, and the tongue is broad, thick, and lengthened. The mucous membranes are also generally hypertrophied. The larynx is often enlarged in all directions, its mucous membranes are thickened, and the voice is hoarse and deep. The lymph-glands of the neck may also enlarge.

FIG. 291.



Shape of the head in acromegaly. (After Schultze.)

Struma may be present, but it is more common to find the thyroid gland smaller than normal. The thickened, bloated skin rarely has a myxedematous character. Fibroma, neuroma, and keloids (Schultze) are often found. The internal organs may also take part in the hypertrophy and enlargement (splanchnomegaly). This is particularly true of the heart, as a result of which cardiac disturbances are often present.

A condition similar to arthritis deformans is occasionally found in the joints. Many individuals affected with acromegaly are giants in stature.

The general condition of the patient also suffers; movements are slow and awkward; the patient is *apathetic, without energy,* and *sleepy; psychic disturbances* occur occasionally. Cephalalgia is a frequent symptom.

Other symptoms of a *cerebral tumor* also occur, though they are not constant. To these belong, according to the observations of Hertel, Uhthoff, and others, *atrophy of the optic nerves, optic neuritis,* and especially ocular disorders of the type of homogeneous *hemianopsia*, hemi-achromatopsia, unilateral blindness with hemianopsia of the other eye, etc.; also *paralysis of the ocular muscles* and *hemianopic pupillary rigidity*. Exophthalmos was observed in one case.

Bulimia, *polydipsia*, and *polyuria* are frequently observed. Mellituria and true diabetes are by no means infrequent. Strümpell mentions an alimentary glycosuria as a symptom.

The movements of the trunk and extremities are generally not involved, though a certain weakness and heaviness may be noticed. The tendon reflexes are normal or increased, or they may be absent.

The disease takes, as a rule, a chronic course and may last for years; an acute onset is, however, not so rare. Relapses may also occur.

Marasmus, diabetes, cardiac lesions, or intercurrent diseases (symptoms of cerebral tumor) are the causes of death.

The pathologico-anatomic examination reveals many alterations. Some of them have been shown on living subjects, with the help of X-rays, almost with anatomic distinctness.

The bones are thickened and deformed partly by an increase in size of some parts, partly by increased depth of the fissures for the blood-vessels and by increased roughness at the points of insertion of tendons and ligaments. Massive stalactite-like osteophytic formations do not occur, though numerous small exostoses are found on the cranium and vertebral bodies. The muscle-insertions are very prominent, and the intercostal spaces widened. The hypertrophy of the bones of the lower jaw is the most marked.

The diseased bones show a thickened periosteum, periosteal new-formations, and thickening of the tendinous and aponeurotic insertions.

Conditions similar to arthritis deformans are found in the joints. The skin is hypertrophied, also the connective tissue of the glands, arteries, and nerve-sheaths (Marie and Marinesco, Arnold). The spinal ganglia and the sympathetic were found hypertrophied several times.

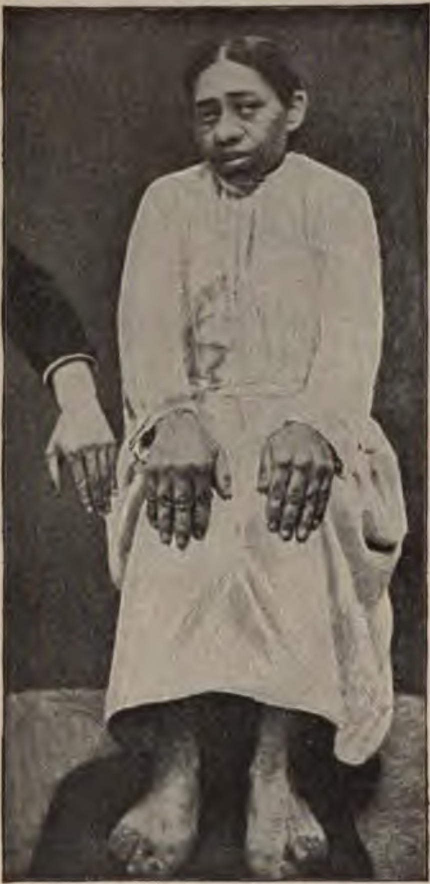
The brain, eyeballs, and abdominal organs may also be increased in size. An asymmetrical degeneration of the posterior columns of the spinal cord was found several times (Arnold, Dallemagne). The *thymus* is often *persistent*, at times hyperplastic.

The thyroids were generally found diseased, either small and atrophic or goitrous.

The most constant condition observed, however, is the *enlarged hypophysis* (Marie). It is hypertrophic and swollen, and tumors of the type of adenoma, sarcoma, and glioma have been described as occurring in it. Hypertrophy of this organ produces a compression of neighboring parts of the brain and cranial nerves, as well as a wearing away of the sella turcica and the sphenoid bone, even to such a degree that the tumor protrudes beneath the pharyngeal mucous membrane.

The diagnosis is easily made in developed cases. Its close relations to myxedema and cretinism may occasionally make a diagnosis difficult, though in acromegaly the myxedematous swelling, coldness and dryness of the skin, loss of hair, etc., are absent; while, on the other hand, hypertrophy of the bones does not occur in myxedema. The two conditions may, however, exist side by side.

FIG. 292.



Woman with acromegaly. (After Sternberg.)

Giantism occurs in about one-fifth of all acromegalics. Sternberg has shown that most of the giants described in literature were acromegalics. It has been assumed that these conditions are identical, that giantism is the acromegaly of the young (Klebs, Massalongo, Brissaud, and Meige). Giantism may, however, occur under other conditions, may be combined with various constitutional disorders, and only forms a predisposition towards acromegaly (Sternberg).

Partial giantism, partial macrosomia,—*i.e.*, hypertrophy of individual parts of the body (macrocheiria, macropodia, hemihypertrophy, etc.),—is distinguished by its congenital origin, by its being confined to one part of the body, and by the extraordinary deformity of the affected limb.

Circumscribed lipoma and *elephantiasis*, or both conditions together, may also produce a kind of partial giantism. Schlesinger describes a partial macrosomia with bulbar symptoms.

Osteitis deformans (Paget) is allied to osteomalacia. Its chief symptoms are a marked distortion of the extremities, especially of the leg, while the hands and feet are spared; the cranium is also increased in volume, though the acromegalic alterations of the soft tissues, etc., are absent. Cerebral symptoms also occur due to compression of the cranial nerves. The disease almost always occurs after the fiftieth year.

Diffuse hyperostosis is a disease of youth, especially characterized by an increase in size of all the cranial bones. The contraction of the cranial fossæ, orbits, and all foramina may cause blindness, deafness, exophthalmos, headache, dementia, etc. This disease, of which little is known, is very rare.

Leontiasis ossea is a tumor-like hyperostosis of the cranium, characterized by the formation of bony tumors. As Starr claimed, many different forms are grouped under this name. He speaks of a case described by me, in which both the bones and the soft structures of the cranium were increased in volume, as one of *megalocephaly*.

True elephantiasis, pachydermia as a result of local asphyxia, and the adipositas dolorosa of Dercum are difficult to distinguish from acromegaly.

Syringomyelia may produce an enlargement of the hands, etc., but the cardinal symptoms of this disease render an error in diagnosis impossible.

Finally, Marie distinguishes a *hypertrophic osteo-arthropathy* (*secondary hypertrophic osteitis* according to Arnold) and gives the following differential points.

In the osteo-arthropathy, or osteopathy, the hands are large and paw-like; the end phalanges appear drum-shaped, and the nails are thick, short, brittle, and bent. In acromegaly the soft structures are particularly involved; in osteopathy it is the bones which suffer the most damage. In acromegaly the region of the carpometacarpal joints is swollen and thickened; while this region is generally spared in osteopathy. The lower jaw is always involved in acromegaly, the vertebræ are thickened, and cervicodorsal kyphosis is present; while in the other disease the lower jaw is not affected, and if kyphosis occurs it is found in the lower part of the vertebral column.

Osteo-arthropathy occurs particularly in combination with cardiac and pulmonary disorders.

The cause and the primary seat of acromegaly are not known. Many facts seem to indicate that the basis of the disease is the alteration in the hypophysis. Experimental observations by Vassale and Sacchi seem to show that the hypophysis is necessary to the organism, and furnishes a specific product to the blood.

Tumors of this gland may, however, exist without acromegaly occurring. Some authors, as Strümpell and Arnold, regard the increase in size of the hypophysis as merely a co-ordinate phenomenon. The part which the thyroid plays in this disease is also obscure. Atrophy of the thyroid may produce a secondary hypertrophy of the hypophysis (von Stieda, Eiselsberg, Hofmeister).

Treatment.—We are still experimenting in the treatment of acromegaly. Thyroid and pituitary extracts have been administered without any noticeable or equable results (Putnam, Bramwell, Ransom, etc.). Caton and Paul sought directly to extirpate the tumor of the hypophysis. Death resulted in three months.

Iodine and mercury are said to have done good in several cases.

SECTION VI.

CONDITIONS OF INTOXICATION, WITH ESPECIAL INVOLVEMENT OF THE NERVOUS SYSTEM.

ALCOHOLISM.

ACUTE alcoholism will not be discussed here. The chronic misuse of alcohol affects injuriously the central as well as the peripheral nervous system. The cerebral functions are variously disturbed. Irritability, indolence, apathy, weakness of the will, loss of the ethical sense and of the memory, are the common psychic conditions produced by alcoholism. Complete *dementia* is not rarely seen in the advanced stage, which may last for years and pass to idiocy. The *acute psychoses* which develop during alcoholism will not be discussed here; only the acute toxic psychosis, κατ' ἐξοχήν, delirium, will be considered.

Chronic alcoholic intoxication also produces cerebral disorders whose symptomatology in many respects corresponds to the picture of the neuroses,—epilepsy, hysteria, and neurasthenia. About thirty per cent. of the alcoholics who are received in the delirium ward of the Charité, in Berlin, are epileptics. They differ from true epileptics in the late development of the disease and its retrogression with the withdrawal of alcohol. The attacks also generally occur as a result of marked excesses. Some authors (Féré, Magnan, Warthmann) look upon alcoholism only as an exciting cause of epilepsy, though this conception does not seem to me to correspond with the facts. Delirium tremens is often ushered in by an attack of epilepsy.

The *subjective disorders* of alcoholics are very similar to those of hysteria and neurasthenia. They often complain of fear, general weakness, sleeplessness, unrest, trembling, depression, pains, loss of appetite, palpitation of the heart, etc. As *objective* symptoms we find *increased knee-reflexes*, increased *mechanical excitability of the muscles and nerves*, *hyperidrosis*, *hypesthesia* and *anesthesia* of the special senses, *vaso-motor disorders*, etc., symptoms which are also similar to those of neurasthenia and hysteria. The attacks of spasms are also very similar to those of hysteria. Unilateral convulsions and paralyses also occur, which are capable of retrogression, and which, as far as my experience goes, are not produced by anatomic alterations. Tetanoid spasms with intact consciousness, similar to attacks of strychnine poisoning, have been described by Siemerling.

The *alcoholic tremor*, one of the most common symptoms, is generally more intense and coarser than the tremor of the neuroses, is found in the lips and tongue as well as in the fingers, and is most intense in the mornings as long as the patient is temperate. It accompanies the voluntary movements without being strictly combined with them.

The *gastric disturbances* are generally due to a true *gastritis*, though they may be merely nervous in character. The *morning vomiting*, nausea and vomiting of slimy masses, and *anorexia* are especially characteristic.

Ocular disturbances, which are very common, may be functional in character (concentric contraction of the visual fields) or due to an *optic neuritis* or a *partial atrophy of the optic nerve* with pallor of the temporal halves (as has been particularly described by Uhthoff). The symptom generally accompanying this is a *central scotoma* for colors (red and green, see page 449).

Another set of symptoms occurring in alcoholism are due to a *peripheral neuritis* caused by the intoxication. Slight degrees are at any rate very common, and probably produce the *cutaneous* and *muscular hyperesthesia*, the *pains* in the legs, and the objective sensory disorders which are found in the area innervated by a cutaneous nerve,—*e.g.*, the external femoral cutaneous. Under the influence of certain factors, this, to a certain extent, latent neuritis may increase until a true alcoholic multiple neuritis is present (which see).

The results of alcoholism described may be variously combined and, in addition, the alcohol may act upon the *blood-vessels, liver, kidneys*, etc., and combinations of these different disorders may produce a varied symptom-complex.

The severe forms of alcoholism are especially noticeable in *whiskey* and *gin drinkers*, though imbibers of wine and beer are not exempt, some results, as multiple neuritis, being particularly common in beer drinkers.

No age is spared, although, of course, most cases occur in middle-aged persons. Individual peculiarities have much to do with the resistance to alcohol, some being able to stand much more than others. Traumata lessen this resistance.

Dipsomania is often a symptom of a *neuropathic disposition*, and such persons are particularly liable to the alcoholic psychoses.

Delirium tremens deserves special mention. It may occur at any time in the course of an attack of alcoholism and often undergoes remissions. Cases are known in which the same person had twenty to twenty-seven attacks of delirium tremens. It has been wrongfully assumed that abstinence in one who was previously a heavy drinker may evoke it. It never occurs without a reason, and is generally brought on by some exciting cause. Severe alcoholic excesses, traumata, and pyrexial diseases (especially pneumonia) are such exciting causes.

The first symptoms are *loss of appetite*, marked tremor, motor and psychic unrest, and depression. The *sleep* next suffers; it is disturbed by fearful dreams; such persons particularly find it difficult to go to sleep, as the mere closure of the eyes evokes frightful visions. The speech is influenced so that it resembles that of paresis, a tremor of the lips and a kind of syllable-stumbling being present. The delirium asserts itself in that the motor and psychic unrest and the tremor increase in intensity, and innumerable *hallucinations* and *illusions* force themselves upon the patient. The hallucinations and illusions are generally connected with real impressions. In the threads, etc., of the bed-cover, he sees lice and spiders, which run around; the figures in the carpet are similarly denoted. At first, when he opens his eyes, he is conscious of being in error, but he is soon mastered by the hallucinations and delusions. The patient sees animals and bugs on the bedclothes, hears unpleasant noises, is completely confused, the phantasies aroused by the hallucinations rapidly following one another. In addition, we find rapid involuntary movements, the patient waving his hands in all directions, pulling at the bedclothes, catching at everything he comes in contact with, etc. He seeks to drive away animals or to catch them; he tries to pull worms, threads, etc., from his mouth; he seeks to guard himself from persons who threaten to kill or poison him, etc. These notions vary rapidly, but are almost always comic, grotesque, or terrifying. Even though terror and anxiety dominate him, he seems generally in particularly good spirits. The patient cannot be aroused from his delirium or can be diverted only for a few moments, in which, however, he is generally docile and not aggressive.

During the delirium the appetite is lost, the pulse is accelerated, reaching one hundred and twenty to one hundred and fifty beats, may be dicrotic, and the temperature may go as high as 104° to 106° F. Elzholz found increase of the leucocytes and excess of polynuclear neutrophile cells over the mononuclear in the blood at the height of the delirium.

The patient is insensitive to pain and moves his injured limbs. For this reason *pneumonia* is often not detected, he neither complaining of pain nor breathing superficially.

The patient perspires rather freely, and the urine of fifty per cent. of the cases contains some *albumin*. Albumosuria occurs more rarely

(Liepmann). He may pass his urine or feces in the bed, but retention of the urine does not occur. The delirium reaches its height in two or three days, lasts three to six days, and generally ends in a deep six to twelve hours' sleep. The pulse again becomes full and slow and the temperature normal. Tremor and unrest may persist for some time afterwards, and a dreamy and partial recollection of the delirium may be present. In less favorable cases it persists and only a new sleep brings recovery.

Some cases remain delirious and die from *collapse*. The pulse becomes smaller and smaller; the temperature remains high or suddenly rises. *Cardiac weakness*, pneumonia, nephritis, trauma, etc., are unfavorable to recovery. It is a bad sign if, after the critical sleep, the pulse-beat and temperature do not go down.

The delirium may be accompanied by hallucinatory confusion, by insanity with delusions of jealousy, or by dementia. Krukenberg has shown that often during the delirium, and even eight to fourteen days after, a concentric contraction of the visual field is present. It also appears as if in rare cases cerebrocortical symptoms may develop at the height of the delirium (Bonhöffer).

The *anatomic alterations* which have been found in the central nervous system in alcoholics are slight, and are limited to hyperemia of the cerebral membranes, slight cloudiness, perhaps edema, of the arachnoid and pia, and external hydrocephalus. *Pachymeningitis hæmorrhagica* is not rarely found. Berkeley has lately, by the newer (microscopic) methods, discovered alterations of the nerve-cells of the brain; Bonhöffer, with Marchi's method, a medullary degeneration of the radiating fibres of the motor cortex. The value and significance of these discoveries are still under discussion.

Treatment.—Withdrawal of all *alcohol*, which is the principal procedure in the treatment, can be successfully carried out only by *hospital treatment*. The necessity for total abstinence in these cases is now almost universally recognized. The institutions of Dr. Smith in Marbach, on the Bodensee, of Dr. Schmitz, in Bonn, and of Dr. Fürer in Rockenau, are for alcoholics, though they are treated in most nervous institutions and sanatoria. Asylums conducted by laymen are those of Ellikon near Zürich, Lintorf near Düsseldorf, Leipe in Silesia, and others. Forel, Kraepelin, and others have lately also advocated the absolute withdrawal of alcohol and continued abstinence. After being discharged the patient should take the pledge or join some temperance society and refrain from drinking for life.

Forel, Ouspensky, etc., report cures through hypnosis. Its value is, however, still *sub judice*.

It has been recommended to excite antipathy for alcohol by adding to all intoxicants strychnine or wine of antimony (Hughes).

Withdrawal of alcohol has often caused the most severe symptoms, as the dementia, to disappear.

Only when pneumonia is present, or collapse is threatened, should alcohol be given. Forel, however, insists that it should not be given even in these contingencies. A strengthening diet is necessary. The delirious patients must be treated in institutions where *isolation* with all precautionary measures can be instituted, and where strait-jackets and similar procedures can be avoided. In mild delirium the *bromides* often are of good service. Narcotics should be used very cautiously; *chloral hydrate* should be preferred to the opiates, though sleeping potions should be used only at long intervals and with careful observation of the heart's action. *Strychnine* has been recommended for the alcoholic tremor. According to my experience the bromides act favorably upon this tremor.

Hyoscine often does good in these cases.

CHRONIC MORPHINE INTOXICATION (MORPHINOMANIA).

By morphinomania Levinstein characterized that *unfortunate condition* in which individuals feel compelled to use morphine as a stimulant and means of enjoyment to such a degree that subjective and objective disturbances of the system are produced, and, in addition, the *diseased condition* which results from its improper use. This condition is almost entirely due to subcutaneous injections of morphine, very rarely to its use per os. The manner in which the trouble develops is generally the following: Originally, the morphine was used to combat some psychic or physical pain. From its continued regular use the body becomes accustomed to it, the desire makes the habitué a slave to the drug, and, as the original dosage is no longer able to produce a condition of euphoria, it is gradually increased. Finally, the individual becomes incapable of any mental work, or of pursuing his calling, or of associating with other people unless he previously takes that quantity of morphine which places him in mental and physical vigor.

With this hunger for morphine, other general disturbances occur (tremor, gastric disorders, cough, etc.), which disappear with the ingestion of the poison.

We cannot speak of morphinomania in those cases in which morphine has been taken regularly for weeks or months on account of an acute painful disease, and is discontinued after the disease is over, even though signs of chronic morphine poisoning may be present.

Physicians are most often attacked by this disorder. Among two hundred and fifty morphinomaniacs ninety-three were physicians; among one hundred male patients forty-two were physicians. *Druggists* are next in point of frequency. It seems to me that this ailment has become less frequent in the last ten years.

A large percentage of those attacked do not have a chronic incurable disease combined with pain; neurasthenics, hypochondriacs, and those with slight depression of the spirits are more often the ones who use or desire the physician to give them morphine.

Naturally, only those become morphinomaniacs in whom the drug produces a *feeling of euphoria*, or a kind of inebriation of the senses combined with much pleasure. As soon as this intoxication disappears and the ordinary depression or pain returns, the desire or feeling of necessity for morphine manifests itself anew; and as such persons are originally weak in will-power, or have become so from the persistent use of morphine, they fall deeper and deeper into its toils. Physicians who, on account of their profession, are compelled to be constantly at their post, and who have constantly this drug at hand, are particularly liable to be overcome.

The dose which is injected is on an average about fifteen grains a day, though cases are known in which forty-five to seventy-five grains were injected. Some of these individuals use another drug also (cocaine, chloral hydrate, chloroform, ether, etc.).

Sooner or later, sometimes in from six to eight months, at times only after a year or so, the *symptoms of chronic morphine intoxication* appear.

Nutrition suffers, the fatty tissue gradually disappears, the skin becomes flaccid and loose, the face pale or ashy-gray, or sometimes deeply red. The *pupils* are generally narrow and react slowly to light. Diplopia and *paresis of the accommodation* occur occasionally. *Hoarseness, thirst, loss of appetite, tremor,* and *disturbances of speech, depression,* and *unrest,* inability for mental work, etc., also occur, symptoms which, for a long time, are relieved only by morphine. After the injection the patient feels rejuvenated and capable of doing anything.

The *demoralizing* influence of the morphine habit is well known. The individual is unreliable, untruthful; his ethical sense becomes lost. A true toxic psychosis rarely results. As a rule, *obstipation* develops, and often gastric disturbances. *Impotence* and *amenorrhea* are constant results.

Azoospermia, or absence of spermatozoa in the seminal fluid, has been observed, also *albuminuria.* Conditions of *sleeplessness,* or slight mental confusion, or attacks of unconsciousness, may be transient symptoms.

In a few cases fever has been observed. *Marasmus* finally results, from which the patient dies. *Multiple abscesses* are often observed, produced by the injections, and act unfavorably upon the general condition. Infectious diseases, particularly pneumonia, take an unfavorable course in these subjects.

If the poison is withdrawn from the individual, symptoms arise which are called the *phenomena of abstinence.* A few hours after the last injection the patient becomes *restless, anxious, excited,* and longs for his morphine. He cannot sleep, jumps from his bed, and moves around here and there. *Nausea, vomiting, diarrhea, palpitation of the heart, hyperidrosis, coryza, ptyalism,* yawning, cough, tremor, neuralgic pains, *accommodation paresis,* and dilated, unequal pupils are some of the symptoms which occur soon afterwards. The symptoms gradually grow worse, hallucinatory excitement, mania, and epileptic or choreic attacks occurring occasionally.

Levinstein describes a delirium very similar to that of alcoholism. A stadium may follow this, in which hallucinations, particularly of an hypochondriac character, harass the patient. It lasts only a few days. *Collapse* is the most dangerous symptom of this period, death often resulting in and from it. The pulse becomes suddenly irregular, slow, sinks to forty or even to thirty, the patient becomes unconscious, breathes laboriously, the face becomes pallid and sunken, etc. The sexual desires are aroused in the first days of abstinence.

Prognosis.—The prognosis is very unfavorable. Even if the withdrawal of the drug is accomplished, the danger of a relapse is very great. Of eighty-two patients of Levinstein, all males, sixty-one relapsed; of twenty-eight females, ten; of thirty-two physicians, twenty-six. The best prognosis is in those patients who use the morphine for a disease which has become cured in the mean time.

Treatment.—*Prophylaxis* is of the utmost importance. Careless use of morphine and of opiates cannot be too strictly avoided. The physician should never allow others to use the hypodermic syringe except in cases where death is expected. If the physician finds it necessary to give morphine, he should stop its use as soon as possible. The greater the euphoria produced by the morphine the more caution is necessary in its use.

The only treatment of morphinomania is by the withdrawal of the drug, even though definite results have been secured in only a few instances. Obersteiner, however, is not right in limiting the conditions necessary for this plan of treatment. Levinstein was in favor of a *sudden* withdrawal, and used a modified method only under certain circumstances,—for instance, when the morphinomania was combined with grave diseases of the internal organs and when collapse was threatened. In the modified method the full dose is given, then an aliquot part given for a few days. Other physicians who have had experience in these cases (Burkardt, Müller, Obersteiner, etc.) are in favor of a *gradual withdrawal.*

Withdrawal is practicable only in an institution where the patient can be constantly and carefully watched. I have come across but three or four cases in my practice who were able to quit the use of this drug of their own accord.

Sometimes a desire for suicide becomes noticeable, which demands particularly careful watching.

Great stress must be laid during this withdrawal upon keeping up the strength of the patient, and in this respect alcohol (champagne, port wine, cognac, etc.) must be used, though as sparingly as possible. Various drugs have been recommended to replace the morphine, as cocaine, sparteine, nitroglycerin, etc. Cocaine should not, however, be used. It renders the withdrawal of the morphine easy, but produces severe psychic disorders, the *cocaine psychosis*, which is a particularly varied form of hallucinatory paranoia. The use of cocaine often converts a morphinomaniac into a "cocaine fiend."

Chloral hydrate may cause delirium. Codeine, belladonna, the bromides, quinine, etc., may be tried, but should be used only for a short time.

Baths with cold douches are beneficial; general massage may also be used for the long, persistent insomnia. Hypnosis might be tried for the same purpose.

Severe conditions of collapse are combated most easily by morphine; generally only a small dosage (.03) is necessary, which, however, if the desired effect has not been secured, must be repeated.

After the drug has been withdrawn, the patient must still be watched over by the physician, and should not be permitted immediately to return to his family and work. It is especially advisable to have him take a sea-journey afterwards, the ship's doctor being carefully instructed about the case. I secured a permanent recovery in one case by this method. In another case, the patient, after reaching Helgoland to winter, turned to chloroform as a tipple, and this was probably the cause of his death, which occurred there.

Above everything else, avoid at any time giving a patient who is cured an injection of morphine. Even when the individual undergoes a remission, the withdrawal has some effect, as from that time smaller doses are required.

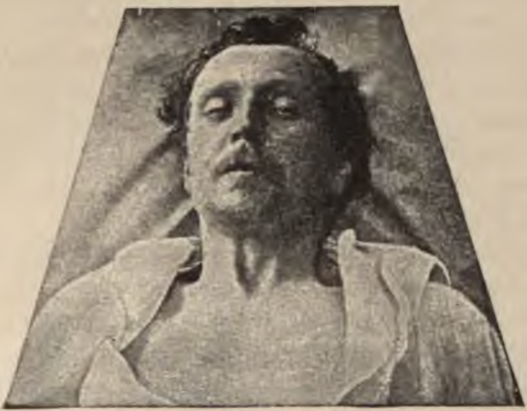
CHRONIC LEAD-INTOXICATION has been described on page 318.

TETANUS.

The most important, perhaps the only, cause of tetanus is an infection by the bacillus tetani, which was discovered by Nicolaier. Kitasato has made pure cultures of it. This bacillus, found in the earth, enters the body through a wound of the skin, or by way of the mucous membranes (pharynx, uterus), and produces local and constitutional disease. It is not the bacillus itself, but its products—tetanin, tetanotoxin, or spasmotoxin, and a very poisonous toxalbumin obtained from pure cultures—which produce tetanus. A laboratory infection resulting in this way has recently been described.

Dirty wounds—either from dirt, splinters of wood, or other foreign bodies (particularly horses' excrement)—are the starting-places for the disease. The spores of the bacillus may remain on foreign bodies virulent for years. *Puerperal tetanus* is, without doubt, also of a similar origin, as is also *tetanus neonatorum*. The patient may be infected through a tampon or from the forceps in the first case, and through the navel in the other. An *idiopathic* or *rheumatic tetanus*, so often spoken about, does not occur; in these cases the wound has been overlooked. *Endemic* or *epidemic* attacks of tetanus have been repeatedly observed. It is also certain that the disease occurs more frequently in the tropics than with us.

FIG. 293.



Facial expression and posture in tetanus. (After Brunner.)

Symptomatology.—As a rule, the initial symptoms do not occur until from five to ten days after the injury, sometimes longer, but in some cases may come on within a shorter period.

Almost always the first thing noticed is a *contraction* of the muscles of the jaw and neck, which may be so slight that a diagnosis of rheumatism is made. It soon afterwards, however, *increases* to a tonic spasm.

Trismus is almost always the first symptom, and soon afterwards *opisthotonos* develops. The *pharyngeal* muscles may become involved early; a feeling of tension in the throat or a *difficulty in swallowing* will then be among the early symptoms. The intensity of the spasm increases from day to day, extends to the *face*, *trunk*, and finally to the *extremities*, though the arm, especially in its distal part, may be spared. In this descending progression of spasm from muscle-group to muscle-group, a certain order of development is noticed. In some cases the ocular muscles are also affected.

The condition of the patient becomes gradually worse, the more so as the *respiratory* muscles become involved in the spasm. He lies in bed, breathing heavily, with rigidly contracted features, the brow corrugated, the eyelids drawn together from spasm of the orbicularis, the angle of the mouth drawn downward (see Fig. 293), the jaws tightly pressed together, the head drawn backward, the abdominal muscles as hard as a board, and the muscular contour of the extended legs sharply mapped out. The muscles relax only in sleep. In a few cases emprosthotonos or pleurothotonos occurs instead of opisthotonos. This chronic spasm causes a certain degree of pain, often severe, which increases during the attacks.

From time to time a *convulsive increase* of the muscular tension occurs, which lasts a few seconds and causes excruciating pain. These attacks, which last only a few seconds, may be produced by *reflex action*, —handling or shaking the patient or irritating his special senses. *Dyspnea* may result from involvement of the laryngeal and thoracic muscles. Speech and deglutition are generally hindered, as is also the evacuation of the bladder and bowels. Complete retention of the urine may take place. No disorder of sensibility or of the mind occurs, the patient being perfectly conscious. The temperature is normal or increases when death approaches, sometimes to a considerable degree. A post-mortem increase to over 112° F. has been observed. Pronounced sweating is an almost constant symptom. Corresponding to it we find the quantity of urine decreased and strongly concentrated. Albuminuria has been observed. The local muscular spasms so often seen in experimental tetanus are rarely observed in man.

In a typical case Rose would differentiate five stages: (1) that of trismus; (2) that of general rigidity; (3) that of the general convulsions; (4) that of reflex convulsions; (5) that of exhaustion. Some have also classified tetanus into acute, chronic, mild, immediate tetanus, etc., according to the acuity of development, course, and severity of the symptoms (Larrey, Rose).

The disease generally lasts for several weeks. If it takes a favorable course, the attacks disappear, then the continuous contraction lessens, and the trismus ceases. Death results in about ninety per cent. of the cases in five or six days, though it may not occur until fourteen days. *Asphyxia*, *exhaustion*, or *cardiac paralysis* produces a lethal result. After the first week recovery is probable; after fourteen days, almost certain. If the symptoms are mild from the first, the prognosis is good. If they are severe or the time of incubation is short (from four to five days), the prognosis is bad.

The diagnosis is not difficult. Tetanus may, however, be at first confused with *strychnine poisoning* or with *hysteric opisthotonos*. Strychnine poisoning may be differentiated by the history, the rapid onset of the symptoms after the poisoning, the immediate wide extension of the contractions, and the prominent reflex spasms. It may be distinguished from hydrophobia by the permanent rigidity.

Hysteric opisthotonos is rarely combined with trismus, and the other phenomena of hysteria are generally detectable. *Anatomic alterations* have been found, but not constantly enough to be of any moment. These were neuritic processes, alterations of the ganglion-cells of the anterior horns (in experimental tetanus by Goldscheider and Flatau and Beck). It may be assumed as certain that the disease is due to a toxic injury of the gray matter of the cord and medulla, though a direct lesion of the peripheral part also occurs.

Treatment.—Avoidance of all excitement. The patient should be placed in a dark, noiseless room, the bed should not be shaken, and all manipulations of his body are contra-indicated. A strengthening diet is necessary; the tube can be passed through his nose or between the teeth, or nutritive clysters may be employed. Chloral hydrate should be used to combat the muscular contractions. It may be given alone or with morphine, administering about one hundred and eighty to two hundred and twenty-five grains a day, up to forty-five grains at a dose. I have seen three of the four cases treated by me cured in this way. Others prefer chloroform. Amyl nitrite, Calabar bean, and curare are also recommended. *Warm baths* are of benefit. Asphyxia must be combated by artificial respiration. An amputation on account of tetanus is never advisable. The wound should be cleansed and disinfected; caustics and the cautery are also in place. If it is confirmed that the poison is excreted by the kidneys, the use of diuretics is certainly indicated.

Recent discoveries have led to newer methods of treatment. Behring showed that the blood-serum obtained from animals immunized against tetanus is not only immunizing but also curative. The manner of immunization cannot be discussed here. From this curative serum, an *antitoxin* has been prepared. Behring and Knorr, as also Tizzoni, have made preparations for immunization and for the treatment of this disease. The results obtained from its use have not been sufficient to permit a decisive judgment of its efficacy. They thus far seem to indicate that the other methods of treatment must not be neglected, and that in developed cases it is disappointing (Sahli, Rose).

Veterinary surgeons also speak with reserve of the benefit of this treatment.

Prophylaxis is very important in tetanus, as careful antisepsis will often prevent its occurrence.

CEPHALIC TETANUS; HYDROPHOBIC TETANUS (ROSE); PARALYTIC TETANUS (KLEMM); BULBAR TETANUS (JANIN).—This is probably merely a variety of general tetanus. The tetanus bacillus has several times been found in the pus of a wound, but is often missed, while the secretion, blood, and transudate in inoculations reveal its nature.

It results after injuries of the *face* and *cranium*, especially in wounds of the orbit and bridge of the nose. It may also arise from a carious tooth, an otitis, etc. The incubation period is eight to nine days. A *spasm* of the facial upon the site of the injury is first noticed. ***Trismus*** next results, first on one side, then on both. The muscular contraction spreads to the pharyngeal and laryngeal muscles on the one hand, and to the muscles of the neck on the other hand. Soon thereafter the facial spasm changes into a facial paralysis, or this occurs immediately without any previous spasm. In some cases the ocular muscles on the same side become affected. If the wound is in the middle line the symptoms may occur bilaterally (Brunner). A *reflex* irritability of the pharynx is generally present, so that attempts to eat cause a *deglutitory spasm.* This symptom has led to the name hydrophobic tetanus being given to this disease. Spasms of the respiratory muscles also may occur. A slight *hypesthesia,* also hyperesthesia, has been several times noticed in the face. The electric excitability remains normal or is somewhat increased.

The muscular rigidity may spread downward or remain stationary. In all other respects the symptomatology is similar to that of tetanus. Fever and acceleration of the pulse may occur with an acute course.

The prognosis is grave, though better than in general tetanus. Death generally results; recovery took place in only twenty-four out of fifty-nine cases.

Nerlich found vacuolization of the ganglion cells in the motor fifth nucleus, also in the nuclei of the facial and hypoglossus, and regarded these as the anatomic basis of the disease. Others report negative results. Alterations in the facial nerve have not been found.

The treatment is identical with that for tetanus. In a few cases (Trapp, Helferich), tetanus antitoxin is said to have produced a cure.

INDEX

A

Abasia, 730
Abdominal muscles, function of, 36
  reflex, 65
Abducens, nucleus of, 420
Abscess of the brain, 545
Accessorius, nucleus of, 115
  paralysis of, 306
  spasm of, 794
Achillodynia, 373
Achromatopsia, 670
Acoustic diseases, peripheral, 299
  nucleus, 421
  tracts, 410
Acromegaly, 870
Acroneuroses, 851
Acroparesthesia, 844
Acrotrophoneuroses, 851
Active movements, examination of, 24
Acute anterior poliomyelitis, 152
  ascending paralysis, 332
  ataxia, 207, 213, 313
  bulbar neuritis, 642
    paralysis, 635
  circumscribed edema, 843
  lethal hysteria, 694
Adhesions of the spinal column, 196
Ageusia, 73
Agoraphobia, 726
Agraphia, 455, 457
Akinesia algera, 731
Alcoholic neuritis, 310
Alcoholism, 877
Alexia, 454, 463
Algesimeter, 55
Allesthesia, 61
Allocheiria, 61
Alopecia, 69
Amaurosis, hysteric, 670, 672
Amaurotic family idiocy, 544
Amimia, 455
Ammonis, cornu, sclerosis of, 777
Amyotrophic lateral sclerosis, 163
Analgesia, 60
  in tabes, 124
Anarthria, 90, 455
Anemia, cerebral, 496
  pernicious, spinal-cord lesions in, 148
Anesthesia, hysteric, 668
Aneurisms, miliary, 500
  of the cerebral arteries, 583, 642
Angina pectoris, hysteric, 692
Angioneuroses, 840
Angioneurotic edema, 843
Anisocoria, 84
Ankle-clonus, 22
    false, 22
Anorexia, hysteric, 686
  nervous, 712
Anosmia, 72
Anthropophobia, 726
Anuria, hysteric, 687
Aphagia algera, 733
Aphasia, 90, 454
  amnestic, 456
  motor, 455
  sensory, 455
  treatment of, 467
  visual, 463
Aphonia, hysteric, 683
  spastic, 89, 673
Aphthongia, 793
Apoplectic attacks, 503
  bulbar paralysis, 635
Apoplectiform seizures in multiple sclerosis, 211
Apoplexia medullæ spinalis, 219
Apoplexy, cerebral, 430, 500
Apraxia algera, 732
Arachnitis gummosa spinalis, 198
Arc de cercle, 675
Archineuron, 106
Argyll-Robertson pupil, 124
Arsenical paralysis, 319
Arteritis, syphilitic, 397
Arthritis deformans of the spinal cord, 193
Arthrogryposis, 812
Arthropathies, dystrophic, 175
  in syringomyelia, 230
  tabetic, 129
Articular diseases in hemiplegia, 439
  neuralgia, 679
Asphyxia, local symmetric, 846
Associative centres, 399
  ocular paralysis, 78
Astasia, 730
Asthenic bulbar paralysis, 648
Asthenopic disorders, 707
Astrophobia, 726
Ataxia, acute, 207
  cerebellar, 39, 625
  gait in, 38, 53
  hereditary, 149
  in cerebral diseases, 405, 445, 625
  in tabes, 125
  static, 38

Ataxic paramyotonia, 181
Athetosis, 440
Athyreosis, 868
Atony, 23
Atrophia muscularis progressiva spinalis, 167
Atrophy in hemiplegia, 439
  in tabes, 127
Auditory centres, 421
  disorders, 299
  examination, 73
  tracts, 410
Auræ, epileptic, 766
Aural vertigo, 760
Autoplastie à lambeaux, 247

B

Baillerger's striæ, 389
Barber's spasm, 811
Basedow's disease, 855
Bastian's hypothesis, 108
Berger's paresthesia, 846
Beriberi, under multiple neuritis, 309
Bladder, general functions of, 109
Blepharospasm, blepharoclonus, 789
Blushing, morbid, 842
Bracelet (Nussbaum's), 810
Brachial neuralgia, 360
Brachialgia, 362
Brachialis internus, function of, 28
Brachium conjunctivum, 407
Bradycardia in neurasthenia, 710
Bradylalia, 90
Brain, anatomy and physiology of, 380
  blood-vessels of, 424
Brodie's symptom, 680
Bromism, 781
Brown-Séquard's paralysis, 120
Bryson's symptom, 859
Bulbar neuritis, 642
  paralysis, acute apopletic, 635
    cerebro-, 644
    from compression, 642
    in amyotrophic lateral sclerosis, 165
    infantile, 634
    progressive, 629
    unilateral, 635, 636
    without any anatomic lesion, 648
  symptoms in gliosis, 231
    in leukemia, 642

C

Cachexia pachydermique, 866
  strumipriva, 867
Caisson disease, 223
Calot's method, 192
Cancrophobia, 736
Canities, 70
Caput obstipum, 795
Carcinoma of the spinal column, 192
Caries of the spinal column, 187
Carrefour sensitif, 467
Catalepsy in hysteria, 776
  of hypnosis, 700
Cataphoresis, 349
Cauda equina, anatomy of, 91
    diseases of, 233
      differential diagnosis between, and diseases of the conus, 235
    injuries of, 234
Causalgia, 59
Cavities in the spinal cord, 227
Central convolutions, 384
  ganglia, 413
  hematomyelia, 220
  tegmentary tract, 405
Centre, ciliospinal, 108
Centres for bladder and rectum, 109
Cephalalgia, cephalæa, 756
Cephalic tetanus, 888
Cerebellar disturbances of gait, 53, 658
  peduncle, 408, 657
Cerebello-olivary tract, 406
Cerebellum, atrophy of, 659
  diseases of, 656
  functions of, 656
Cerebral abscess, 545
  anemia, 496
  convolutions, 383
  cortex, 388
  hemorrhage, 500
  hyperemia, 498
  paralysis of childhood, 532
  peduncles, fibres of, 400
  softening, 517
    chronic progressive, 525
  syphilis, 597
  tumors, 564
Cebrospinal meningitis, epidemic, 483
Chasma, 802
Cheyne-Stokes breathing, 432
Chorea corpuscles, 827
  electric, 830
  hereditary, chronic progressive, 828
  hysteric, 819, 693
  magna, 693
  minor, 819
  nocturna, 823
  of pregnancy, 820, 825
  permanens, 826
  post-hemiplegic, 441
  senilis, 826
Chvostek's symptom, 815
Ciliary neuralgia, 354
Circulatory disturbances in the brain, 558
Clark's columns, 103
Claudication intermittente, 372
Claustrophobia, 726
Claw-hand, *main en griffe*, 30
Clownism in hysteria, 675
Coccygodynia, 376
Collapse in morphinomania, 883

Columnæ vesiculares, 102
Coma, 431
  apoplectic, 504
Combined system disease of the spinal cord, 146
Commissural cells, 105
Commotio spinalis, 186
Compensatory movements, 442
Compression bulbar paralysis, 642
Conducting tracts of the spinal cord, 102–106
Congestion, 498
Conjugate paralysis of ocular muscles, 78
Contracture, active, 37
  hysteric, 679
  paralytic, 36
Conus terminalis, diseases of, 233
    injuries of, 185
Convergence, paralysis of, 81
Convolutions of the brain, 383
Co-ordination, disturbances of, 37
Coprolalia, 803
Corneal reflex, 84
Cornu ammonis, sclerosis of, 777
Corpus restiforme, 408
  trapezoid, 421
Cranial deformities, 18
  examinations, 18
  nerves, 413
    origin of, 77
    peripheral paralysis of, 279
Cranio-cerebral topography, 387
Cranio-neuralgia, 355
Cremasteric reflex, 65
Cretinism, sporadic, 868
Crises, gastric, 128
  laryngeal, 128
Crural paralysis, 264
Crutch-palsy, 249, 264
Cyrtometer, 388
Cysticercus cerebri, 585
  racemosus, 586

D

Dark perimeter, 76
Deafness after cerebrospinal meningitis, 485
  from hysteria, 672
Decrease of electric excitability, 46
Decubitus, acute, 207, 509
  in myelitis, 206
Degeneration, reaction of, 49
    partial, 50
  retrograde, 113
  secondary, 110
  signs of, 18
Deglutitory paralysis, 87
    in hysteria, 684
  spasm, 794
Délire du toucher, 728
Delirium, 431
  epileptic, 770
Delirium, hallucinatory, 665
  tremens, 879
Deltoid, function of, 27
  paralysis of, 27
Démarche tabéto-cérébelleuse, 150
Dementia, paralytic, 611
  senile, 621
Dermatomyositis, 338
Dermography, 709
Deviation, conjugate, 81, 443
Diaphragm, spasm of the, 802
  phenomenon, 253
Diathesis, neuropathic, 15
Diffuse diseases of the spinal cord, 181
  sclerosis of the brain, 216
Diphtheritic paralysis, 321
Diplacusis, 74
Diplegia, facial, 292
  spastic infantile, 533, 541
Diplopia, 79
  monocular, 79
Disseminated sclerosis, 209
Divergence, paralysis of, 81
Diver's disease, 222
  paralysis, 222
Double sensations, 61
    in tabes, 127
Dry mouth, 763
Duboisin in paralysis agitans, 838
Dura mater, inflammations of, 468
Dynamometer, 25
Dysarthria, 90
Dysbasia, 730
Dyslexia, 455
Dysopsia algera, 731
Dyspepsia, nervous, 712
Dysthyreosis, 862
Dystrophy, progressive muscular, 171

E

Echinoccus cerebri, 585
Echolalia, 803
Eclampsia, infantile, 785
  parturient, 786
Elæomyenschisis, 800
Electric examination, 41
  excitability, alterations in, 46
Embolus of the cerebral arteries, 517
Encephalitis, acute hemorrhagic. 526
  acute non-purulent, 526
  purulent, 545
  superior, 529
Encephalomalacia, 517
Encephalometer, 388
Encephalomyelitis, disseminated, 207
Encephalopathy, saturnine, 318
Enuresis, nocturnal, 712
Epicondylalgia, 347
Epidemic rigidity of the neck, 482
Epilepsie marmottante, 771
Epilepsy, 764
  cortical, 433
  in cerebral paralysis of childhood, 540

Epilepsy, minor, 769
    procursive, 769
    senile, late, 765
Epileptic insanity, 771
Equivalents, epileptic, 770
    hemicranial, 753
Erb's paralysis, 256
    point, 141
    symptom, 816
Erections in neurasthenia, 711
Erythromelalgia, 850
Erythromelia, 851
Erythrophobia, 709
Essential hereditary tremor, 67 195
Ether, paralysis of the musculo-spiral from, 267
Exairesis of nerves, 351
Exercise spasm, 812
Exhaustion, anemic conditions of, 237
Exophthalmos, 80
    in Graves's disease, 856
Exothyropexia, 865
Extension-beds of plaster of Paris, 191
Extensors carpi, function of, 29
Extradural paralysis, 553

F

Facial nucleus, 420
    paralysis, 291
    phenomenon, 815
    spasm, 787
Facies, myopathic, 174
Fasciculus, anterior lateral ascending, 97
Fear, conditions of, 725
    in hysteria, 665
    in neurasthenia, 705
Femoral reflex, 66
Fibres of the spinal cord, 97
Flechsig's treatment, 783
Flexibilitas cerea, 676
Flexores digitorum, function of, 30
Flute-players' spasm, 811
Focal symptoms in cerebral disease, 433
Folie du doute, 726
Foot, tremor of, 22
Fractures of the spinal column, 181
Frenkel's treatment of ataxia, 138
Friedmann's symptom-complex, 740
Friedreich's disease, 149

G

Gait, examination of the, 52
Ganglion, Gasserian, extirpation of, 351
Gangrene, symmetric, 846
Gastralgia, 377
Gastralgokenosis, 712
Gastric crises, 128
Gastroxynsis, 713
General symptoms of cerebral diseases, 429
Geniospasm, 789
Gerhardt's signs, 560
Gliosis, spinal, 227
    unilateral, 231
Glisson's capsule, 191
Globus, hysteric, 673
Glossal muscles, paralysis of, 85
        spasm of, 793
Glossodynia, 763
Glossopharyngeal nucleus, 424
    paralysis, 301
    spasm, 794
Glossopharyngolabial paralysis, see Bulbar paralysis
Glossospasm, 793
Glossy skin, 69
Glutei, function of, 32
Glycosuria, alimentary, 743
Goose-skin reflex, 69
Gowers's tract, 97, 405, 104
Graefe's symptom, 857
Graphospasm, 807
Graves's disease, 855
Gray matter of the spinal cord, 94
Greffe nerveuse, 247
Griesinger's signs, 560
Gummata, 597

H

Hallucinatory delirium, 665
Haphalgesia, 671
Head, tetanus of, 888
Headache, 756
    in cerebral diseases, 429
Head's hypothesis, 102
Heart crises, 128
Hematoma, 468
Hematomyelia, 219
Hemianesthesia, crossed, 445
    in cerebral diseases, 444
Hemianopsia, 76, 445
    bilateral, 76
    bitemporal, 450
    bitemporalis fugax, 605
    homonymous, 450
Hemianoptic pupillary rigidity, 451
Hemiataxia, 446
Hemiathetosis, 442, 538
Hemiatrophy, lingual, 85, 308
    progressive facial, 853
Hemichorea, 440
    posthemiplegic, 440, 822
Hemichromatopsia, 451
Hemicrania, 748
    angioparalytica, 750
    angiospastica, 750
    cerebellar, 751
    equivalents of, 753
    ophthalmic, 751
    permanens, 753
Hemihyperesthesia cruciata, 445
Hemihyperidrosis in tabes, 133
    unilateral, 72
Hemihypertrophy, facial, 855

Hemiparetic form of multiple sclerosis, 213
Hemiplegia, 437, 505
  alternans, 443, 624
  cruciata, 627
  homolateral or collateral, 438
  hysteric, 682
  sine materia (without organic basis), 524
  spastic infantile, 532
Hemiplegic contracture, 438
Hemispasm, glossolabial, 682
  glossolabiomaxillary, 682
Hemorrhage, cerebral, 500
Hereditary ataxia, 149
  spinal cord diseases, 202
  syphilitic cerebral diseases, 609
Heredity, 15
Hérédo-ataxie cérébelleuse, 151
Herpes in meningitis, 484
  in neuralgia, 344
  zoster, 363
Heterotopia in the brain, 623
  of the spinal cord, 236
Hoffmann's symptom, 816
Huntington's disease, 828
Hydrocephaloid, 497
Hydrocephalus, acquired, 593
  congenital, 589
  external, 589
  idiopathic, 594
Hydromyelia, 227
Hydrops, intermittent articular, 69, 844
Hydrotherapy in neurasthenia, 720
Hyoscin in paralysis agitans, 838
Hyperacusis, 294
Hyperemia, cerebral, 498
Hyperesthesia, 60
  acoustic, 74
  plantar, 667
Hyperidrosis, 72
  in Graves's disease, 859
Hyperkinesis, 67
Hyperostosis, diffuse, 875
Hyperthyreoidization, 862
Hypnoid conditions in hysteria, 676
Hypnosis, 700
Hypnotism, 700
Hypoglossal nucleus, 424
  paralysis, 308
  spasm, 793
Hypophysis in acromegaly, 873
Hypotony, 23
Hysteria, 661
  acute lethal, 694
Hysteric anesthesia, 668
  contracture, 679
  fever, 688
  paralysis, 681
  spasms, 673
Hystero-epilepsy, 693
Hysterofrenatic points, 667
Hysterogenetic zones, 667

I

Idiomuscular contractions, 52
Idiopathic contractures, 817
  muscle spasms, 812
Ileopsoas, function of, 32
Impotence in neurasthenia, 712
Inanition, delirium of, 497
Incontinence of urine, 109
Incoördination, 38, 125
Infantile cerebellar paralysis, 659
  cerebral paralysis, 532
  spinal paralysis, 143
Influenza-encephalitis, 531
Infraspinatus, function of, 27
Intention tremor, 211
Intercostal neuralgia, 363
Intermediary spasms, 773
Intermittency of morphinomania, 882
Intermittens larvata, 353
Intermittent limping, 372
Internal capsule, 399
Interossei, function of, 30
  paralysis of, 30
Intoxication amblyopia, 450
Inunction cure, 610
Involuntary laughter in bulbar paralysis, 638
    in multiple sclerosis, 211
Irritable breast, 365
Ischemic muscular paralysis, 245
Ischiadicus, paralysis of, 276
Ischuria, parodoxic, 110

J

Jacksonian epilepsy, 433
Jendrassik's reinforcement, 21

K

Keirophobia, 811
Keirospasm, 811
Keratitis, neuroparalytic, 69, 289
Keraunoneuroses, 734
Kernig's symptom, 195, 474
Kinesiesthesiometer, 58
Klumpke's paralysis, 257
Knee phenomenon or reflex, 21
  reflex, decrease of, 107
    increase of, 107
Kubisagara, 762

L

Lagophthalmos, 294
Laminectomy, 187, 192
Lancinating pains, 124
Landry's paralysis, 333
Laryngeal crises, 128
  muscles, 88
  nerves, 302

Laryngeal paralysis, 304
    respiratory, 305
  vertigo, 761
Lasègue's symptom, 371
Lateral sclerosis, 140
    amyotropic, 163
Lateropulsion, 834
Lathyrus poisoning, 143
Latissimus dorsi, function of, 28
Lead paralysis, 315
Lemniscus, chief, 405
  decussation of, 404
  lateral, 410
  middle, 405
Leontiasis ossea, 875
Lepra, differential diagnosis between gliosis and, 232
Leptomeningitis, acute cerebral, 471
  acute spinal, 194
Lethargy, 676
  in hypnosis, 701
Levator anguli scapulæ, function of, 26
    paralysis of, 26
Liquor, cerebrospinal, its condition in disease, 479
Little's disease, 143
Lobar sclerosis, 534
Localization in the cortex, 391
  in the spinal cord, 114
  laws of, 391
Locomotor ataxia, 123
Loose joint in poliomyelitis, 154
Lordosis from muscular paralysis, 36
  of the spinal column from progressive muscular atrophy, 173
Lues, cerebral, 597
  spinal, 198
Lumbar puncture, 478
Luxation of the spinal column, 182
  paralysis, 258

M

Macroesthesia, 61
Macropsia, 685
Macrosomia, 874
Main en griffe, 30
  succulente, 230
Maladie des tics convulsifs, 803
    impulsifs, 803
Malformations of the spinal cord, 236
Malum Cotunnii, 367
  perforans, 69
    in tabes, 129
Mandibular reflex, 23
Mannkopf's symptom, 60
  in traumatic neuroses, 744
Masseter clonus, 23
Masticatory muscles, function of, 85
  paralysis of, 85
  spasm of, 792
Mastodynia, 365
Masturbation, 710
Mechanical excitability of the muscles and nerves, 52
Median, paralysis of, 269
Megalocephaly, 875
Mellituria, 628
Ménière's vertigo, 760
Meningism, 477
Meningitis, acute purulent cerebral, 471
  acute spinal, 194
  basilar chronica simplex, 494
    gummatous, 598
  cerebral, 494
  chronic spinal, 196
  epidemic cerebrospinal, 482
  ossificans, 494
  serous, 553, 594
  simple chronic, 494
  spinal, 194
  syphilitic, 198
  tubercular, 498
Meningocele, 237
Meningococcus intracellularis, 471
Meningoencephalitis, syphilitic, 599
Meningomyelitis, syphilitic, 198
Mental condition, examination of, 17
Meralgia, paresthetic, 276
Meteorism in hysteria, 686
Microgyria, 534, 647
Migraine, 748
  ophthalmic, 751
  ophthalmoplégique, 285
Milkers' cramp, 811
Misophobia, 726
Mogigraphy, 807
Mogiphony, 811
Monoplegia, 435
Morbus Basedowii, 855
  sacer, 764
Morel's ear, 18
Morphinomania, 881
Morton's metatarsalgia, 373
Morvan's disease, 231
Motility, examination of the, 19
Motor centres, 392
  conducting tracts, 399, 627
  focal symptoms, 433
  points, 41
  zone of the brain, 392
Multiple neuritis, 309
  sclerosis, 209
  syphilitic radicular neuritis, 608
Muscle defects, congenital, 157
  paralysis, ischemic, 245
  spasms, localized, 787
  tonus, 20
  transplantation in peripheral paralysis, 247
    in poliomyelitis, 158
Muscles, functions of, 25
Muscular atrophy, arthritic, 176
    hereditary form of, 171
    myopathic, 171
    progressive, 167
    spinal form of, 167
  conditions, 19
  sense, 58
Musculocutaneous, paralysis of, 261

Mutism, hysteric, 683
Myasthenia gravis pseudoparalytica, 648
Mydriasis, 83
Myelitis, 204
  bulbar, 636
  cervical, 206
  disseminated, 206
  dorsal, 205
  lumbar, 206
  transverse, 205
Myelocele, 237
Myoclonus, 68, 806
Myokymia, 40
Myopathy, progressive, 171
Myosis, 83
  in tabes, 127
Myospasia, impulsive, 803
Myotonia, acquired, 181
  congenital, 179
Myotonic reaction, 180
Myxedema, 866

N

Narcolepsy, 677, 771
Narcosis paralysis, 256
Nerve-extraction, 351
Nerve-stretching in neuritis, 252
    in tabes, 138
Nerve-suturing, 247
    secondary, 246
Nerve-tumors, 377
Nervous dyspepsia, 712
  retreats, 721
  weakness, 703
Neuralgia, anoperineal, 376
  brachial, 360
  crural, 367
  general discussion, 342
  intercostal, 362
  ischiadica (sciatica), 367
  lumbar, 366
  occipital, 357
  ophthalmic, 353
  phrenic, 359
  pudendo-hemorrhoidal, 375
  spermatic, 375
  treatment of, 347
  trigeminal, 351
  tympanic, 355
  visceral, 377
Neurasthenia, 703
  cordis, 709
  sexualis, 710
  vasomotor, 710
Neurectomy, 351
Neuritis, 248
  acute bulbar, 642
  alcoholic, 310
  ascending or migratory, 238, 249
  diabetic, 325
  disseminated nodal, 248
  gummatous, of the roots, 200
  interstitial hypertrophic, 179
  mercurial, 320
Neuritis, multiple, 309
  optic, 446
  periaxial segmentary, 249
  puerperal, 325
  retrobulbar, 449
  senile, 325
Neurofibromatosis, general, 378
Neurolization, 242
Neurolysis, 241
Neuroma, 377
Neuromyositis, 340
Neuron, 94
Neuropathic diathesis, 15
Neuropile, 94
Neuroses, 661
  traumatic, 733
Neuro-tabes, peripheral, 312
Neurotic form of progressive muscular atrophy, 177
Neurotomy, 351
Nissl's method, 94
Nitroglycerin in migraine, 755
Nodding spasm, 796
Nosophobia, 705
Nystagmus, 81
  in sclerosis, 211

O

Obstetrical paralysis, 259
Obstipation in neurasthenia, 713
Obturator paralysis, 275
Occipital neuralgia, 357
Occupation pareses, 341
  spasms or neuroses, 807
Ocular migraine, 751
  muscles, associated paralysis of, 78
    centres, in cortex for, 395
    congenital paralysis of, 281
    functions of, 78
    nuclei of, 413
    paralysis of, 78, 80
  nerves, paralysis of, 279
Oculomotor nucleus, 413
  paralysis, 281
    periodic, 284
Oculopupillary fibres, 108, 116
  symptoms in plexus paralysis, 258
Olfactometer, 72
Olfactory centres, 398
  fibres, course of, 286
Oliguria, 687
Olive, midlayer of, 405
Onanism, 710
Onychogryphosis, 69
Operculum, 384
Ophthalmoplegia, 652
  congenital, 654
Optic atrophy, 446
    in tabes, 127
  radiations, 411
  tract, 411
Oscedo, 802
Osteitis deformans, 874

Osteo-arthropathy, 875
Osteomalacious paralysis, 238
Osteopathy, 875
Otalgia, 355
Oxyokeia, 294

P

Pachyakria, 870
Pachymeningitis cervicalis hypertrophica, 196
    external, 467
    internal hemorrhagic, 468
    purulent, 553
Palatal muscles, 86
    reflex, 87
Pantophobia, 726
Paradoxic contraction, 66
Parakusis, 74
Paralysis, accessorius, 306
    acute ascending, 332
    acute bulbar, 635
    agitans, 831
    brachial plexus, 258
    circumflex, 261
    crural, 274
    diaphragmatic, 253
    diphtheritic, 321
    examination for, 36
    facial, 291
    glossopharyngeal, 301
    hypoglossal, 307
    hysteric, 681
    long thoracic, 260
    median, 269
    musculocutaneous, 261
    musculospiral, 263
    obstetrical, 259
    obturator, 275
    ocular, 279
    of the insane, 611
    peroneal, 277
    phrenic, 252
    posterior tibial, 278
    posthemiplegic, 442
    progressive glossopharyngolabial, 629
        infantile form of, 634
    saturnine, 315
    sciatic, 276
    spastic, 37
    trigeminal, 286
Paralytic attacks, 617
    contracture, 36
        in infantile paralysis, 153
Paramyoclonus multiplex, 806
Paramyotonia, congenital, 181
Paraphasia, 456
Paraplegia, senile, 209
Parasites of the brain, 585
Parésie analgésique, 231
Paresthesia, general, 61
Parkinson's disease, 831
Passive movements, 20
Patellar clonus, 22
Pavor nocturnus, 707
Pectoralis major, function of, 27
        paralysis of, 27
Pellagra, 149
Periaxial neuritis, 248
Perimetric examination, 75
Perineuritis, 248
Periodic oculomotor paralysis, 284
    paralysis of extremities, 238
Peripheral nerves, diseases of, 239
        traumatic paralysis of, 240
    paralysis, 240
        of spinal nerves, 252
Pernicious anæmia, spinal-cord lesions in, 148
Peroneus, function of, 33
    paralysis of, 33, 276
Personality, double, 678
Perspiratory centres, 71
Perverse sensations, 60
Petit mal, 769
Pflüger's law, 67
Pharyngeal crises, 128
    reflex, 87
Phlebitis obliterans, 200
Phobias, 726
Phobophobia, 726
Phrenic, paralysis of, 252
Piano-players' cramp, 810
Pied tabétique, 129
Plantar reflex, 64
Plaques jaunes, 520
Playfair cure, 723
Plexus, brachial, 254
        paralysis of, 254
    paralysis, lower, 257
        total, 258
        upper, 255
Points, motor, 41
Polio-encephalitis, acute, 529
        inferior, 636
Polio-encephalomyelitis, 531
Poliomyelitis, acute anterior, 152
    of adults, 159
    subacute or chronic, 160
Pollakuria in neurasthenia, 712
Pollutions, 710
Polyclonia, 806
Polyesthesia, 61
Polymyositis, 337
Polyneuritic psychosis, 313
Polyneuritis, 309
    diabetic, 325
    senile, 325
Polyopia, monocular, in hysteria, 685
Pons, diseases of, 623
    hemorrhage of, 509
Porencephaly, 145, 534
Posticus paralysis, 89
Potamophobia, 726
Pott's back, 188
Preachers' hand, 197
Priapism in tabes, 127
Professional pareses, 341
Progressive bulbar paralysis, 629
        infantile form of, 634

Progressive muscular atrophy, 167
        familial type of, 171
        hereditary form of, 171
        infantile, 171
        juvenile, 171
        neural, 177
    paralysis of the insane, 611
Propulsion, 834
Prosopalgia, 351
Prosopoplegia, 291
Prurigo in neurasthenia, 708
Pruritus, 708
Pseudobulbar paralysis, 644
        infantile, 647
Pseudohypertrophy, 171, 175
Pseudomeningitis, 477
Pseudoneuralgia, hysteric, 346
Pseudoneuritis, optic, 447
Pseudoptosis, hysteric, 346
Pseudosclerosis, 215
Pseudotabes, alcoholic, 312
    arsenical, 319
    diabetic, 131, 325
    syphilitic, 202
Pseudotetanus, 817
Pseudotrichinosis, 340
Psychic blindness, 452
Psychotherapy in hysteria, 697
Psychrophore, 718
Ptarmus, 802
Puncture in hydrocephalus, 592, 597
Pupillary inequality, 84
    reaction, 82
        paradoxical, 83
        reflex paths for, 416
    rigidity, 82, 124, 416
Pyramidal decussation, 402
    tract, 399

Q

Quadriceps femoris, function of, 32
        paralysis of, 32
Quadrigeminal diseases, 625

R

Rachischisis, 236
Radiographs in brain-tumors, 579
Railway spine, 236
Raynaud's disease, 846
Recurrent paralysis, 89. See also Vagus paralysis
Reflex areas in the spinal cord, 105
    collaterals, 103
    epilepsy, 765, 771
    neurosis, otitic, 554
    paralyses, 237
    spasms, saltatory, 803
  generation of peripheral nerves, 241
  espiratory spasms, 802
        hysteric, 673
Retrograde degeneration, 113
Retropharyngeal abscess, 188
Retropulsion, 834
Rhenchospasm, 802
Rigidity, congenital spastic, 143
Rinne's test, 74
Romberg's symptom, 125
Ructus, 673

S

Salaam spasms, 796
Saltatory reflex spasms, 803
Sanatoriums, 721
Satyriasis, 127
Sawyers' cramp, 811
Sayre's method in caries, 191
Scanning speech, 90
        in sclerosis, 211
Sciatic phenomenon, 371
Sciatica, 367
    scoliotica, 369
Sclerodactyly, 852
Scleroderma, 851
Sclerosis, amyotrophic lateral, 163
    diffuse, 216, 622, 623
    lobar, 534
    miliary, 623
    multiple, 209
Scoliosis in syringomyelia, 230
Scotoma, 76
    scintillans, 751
Scrotal reflex, 66
Secondary degeneration in brain, 428
        in pyramidal cord, 110
Secretions, anomalies of, 71
Sein hystérique, 666
Senile paraplegia, 209
Sensation, centres for, 405
    disorders of, in brain diseases, 443
    examination of, 54
    partial paralysis of, 61
Senses, examination of the, 72
Sensibilité récurrente, 244
Sensomobility, 125
Sensory disorders, general, 60
    tract, 402
Serratus anticus major, function of, 26
    paralysis of, 26, 261
Sexual neurasthenia, 710
Shock, 236
Singultus, 802
    in hysteria, 673
Sinus thrombosis, 556
Skin, innervation of, 62
    reflexes of, 64
Skull, examination of, 18
Sleeplessness in neurasthenia, 705
    its treatment, 722
Smell, examination of sense of, 72
Sneezing spasm, 802
Solitary fasciculus, 302
        atrophy of, 134
Somnambulism, 677
    of hypnosis, 701
Somnolence, 430
Sopor, 430

- Spasm, facial, 787
  - palatine, 790
- Spasms, 67
  - general, 67
  - hysteric, 673
  - of cervical muscles, 794
  - of extremities and trunk, 800
- Spasmus nutans, 796
- Spastic ataxic paraplegia, 147
  - condition, 37
  - gait, 53
  - spinal paralysis, 140
    - congenital form of, 143
    - hereditary form of, 142, 544
- Speech-centres, 396
- Speech-disturbances, general, 90
  - in cerebral disease, 454
- Spermatorrhea, 711
- Spina bifida, 236
- Spinal cord, 91
    - abscess of, 218
    - arteries of, 122
    - blood-vessels of, 122
    - centres of, 118
    - concussion of, 186
    - glioma of, 222
    - roots of, 114
    - stretching of, in tabes, 138
    - syphilis of, 198
    - tracts of, 97
    - tubercle of, 222
    - tumors of, 222
  - ganglia, 91
  - puncture, 478
- Splanchnomegaly, 872
- Splenius capitis, function of, 35
- Spondylitis, simple, 186
  - tubercular, 187
- Spondylomalacia, traumatic, 186
- Spontaneous amputation, 852
  - fracture in syringomyelia, 230
    - in tabes, 129
  - hemorrhage in hysteria, 687
  - movements in tabes, 126
- Sporadic cretinism, 868
- Staso-basophobia, 731
- Static reflex spasms, 803
- Status epilepticus, 775
- Stellwag's symptom, 857
- Stereognostic sense, 58
- Sternocleidomastoid, function of, 35
- Sternutatio, 802
- Stigmata hereditatis, 18
- Strength of electric current, 45
- Struma in Graves's disease, 855
- Strumectomy in Graves's disease, 865
- Stupor, 430
- Stuttering, 90
  - hysteric, 684
- Suggestion, verbal, 701
- Supinator brevis, function of, 29
  - longus, function of, 28
- Supraorbital neuralgia, 354
- Suprascapular, paralysis of, 362
- Suspension in tabes, 139
- *Suture à distance*, 242
  - *tubulaire*, 242
- Syllable stumbling, 90
- Symmetric gangrene, 846
- Sympathetic, diseases of the, 840
- Syncope, 496
  - local, 847
- Syphilitic cerebral diseases, 597
  - meningitis, 198
  - spinal paralysis, 202
  - vertebral diseases, 193
- Syringomyelia, 227
- System diseases of the spinal cord, 123

T

- Tabes dorsalis, 123
    - course of, 136
    - pathologic anatomy of, 132
    - treatment of, 137
- Tachycardia in Graves's disease, 855
  - in neurasthenia, 709
- Tactile paralysis, 444
- Talalgia, 373
- Tangential fibres, atrophy of, 613
- Tarsalgia, 373
- Taste, examination of sense of, 73
- Taste-fibres, course of, 286
- Tegmentary radiations, 399
- Teleneuron, 106
- Temperature tests, 56
    - after Goldscheider, 56
- Tendon-reflexes, 21
    - increased, 22
- Tendons, plastic operations on, 158, 544
- Tetaniform spasms in brain disease, 435
- Tetanilla, 812
- Tetanus, 885
  - hydrophobic, 888
  - idiopathic, 885
  - neonatorum, 885
  - puerperal, 885
- Tetany, 812
- Thalamus opticus, functions of, 412
- Thermanesthesia, 60
- Thomsen's disease, 179
- Thoracicus longus, paralysis of, 261
- Thrombosis of the cranial sinuses, 556
- Thumb-muscles, function of, 31
- Thyroid, function of, 867
- Thyroidin, 869
- Thyroiodin, 869
- Tibialis anticus, function of, 33
  - posticus, function of, 34
    - paralysis of, 278
- Tic convulsif, 787
  - douloureux, 351
  - général, 803
  - rotatoire, 796
- Tinnitis aurium, 300, 706
- Topoalgia, 59
- Torticollis rheumaticus, 795
- Tracts of the spinal cord, 97

- Transfert in hysteria, 673
  - in neuralgia, 344
- Traumatic hematoma, 470
  - late apoplexy, 470
  - neuroses, 733
  - paralysis of nerves, 240
- Tremor, alcoholic, 878
  - fibrillary, 40, 736
  - general remarks on, 39
  - senile, 837
- Trepanation in brain-tumor, 580
  - in epilepsy, 783
- Triceps, function of, 27
  - paralysis of, 266
  - suræ, function of, 33
- Trichinosis, 340
- Trigeminal neuralgia, 351
  - nucleus, 417
  - paralysis, 286
  - roots, 417
- Triplegia in cerebrospinal lues, 201
- Trismus, 792
  - in tetanus, 886
- Trochlear nucleus, 417
- Trophic centres, trophic nerves, 69
  - disturbances, 69
    - in brain diseases, 446
    - in neuritis, 251
    - in tabes, 129
- Trophoneuroses, 840
- Trousseau's symptom, 814
- Tubercula dolorosa, 378
- Tubercular basilar meningitis, 488
- Tuberous sclerosis, 623
- Tumor cerebelli, 573
  - cerebri, 564
- Tumors of the spinal cord, 222
- Tussis hysterica, 673
- Tympanites in hysteria, 686

U

- Ulcer, perforating, 69
- Ulnar, paralysis of, 271
- Unilateral lesions of spinal cord, 120
- Urethrodynia, 763
- Urticaria factitia, 709
  - interna, 843

V

- Vagus, innervation area of, 302
  - nuclei, 302, 423
  - paralysis of, 302
- Valleix's pressure-points, 344
- Vascular system of the brain, 424
- Vasomotor centres, 68
    - in brain, 628
    - in spinal cord, 102
  - disturbances, 69
    - in brain-diseases, 446
  - neurosis of extremities, 844
  - symptom-complex, 740
- Ventricular hemorrhage, 509
- Vertigo, 759
  - Gerlier's, 762
  - in cerebral disease, 445, 568
  - in neurasthenia, 706
  - Ménière's, 760
  - paralysant, 762
- Vicq d'Azyr's striæ, 389
- Vigouroux's symptoms, 859
- Vision, field of, 74
- Visual aphasia, 463
  - centres, 411
  - field, contraction of, 75
    - examination of, 74
  - tracts, 411
- Vocal cords, hysteric spasm of, 673
    - paralysis of, 88
      - cortical, 395
- Vomiting, hysteric, 686
  - morning, 878

W

- Weber's test, 74
- Weir Mitchell cure, 723
- Westphal-Edinger's nucleus, 414
- Westphal's symptom, 24
    - conditions necessary for its presence, 107
    - in tabes, 124
- Word-blindness, 457
- Word-deafness, 456
- Working ability in traumatic neuroses, 746
- Wrist-clonus, 23
- Writers' cramp, 807

X

- Xerostomia, 764
- Xyrospasm, 811

Y

- Yawning spasm, 802

Z

- Zoster, ophthalmic, 289

THE END.

LANE MEDICAL LIBRARY
300 PASTEUR DRIVE
PALO ALTO, CALIFORNIA 94304

Ignorance of Library's rules does not exempt violators from penalties.

50M-10-63-5632

LANE MEDICAL LIBRARY
STANFORD UNIVERSITY
MEDICAL CENTER
STANFORD, CALIF. 94305

LANE MEDICAL LIBRARY
300 PASTEUR DRIVE
PALO ALTO, CALIF. 94304

L341 Oppenheim, H.
062m Dis. of nervous system
1900 6749

NAME	DATE DUE
M. Cook	OCT 13 1919 Sept 9-19
A. J. Hood	JAN 29 1920
Dr. Rixford	JUN 20 1922
(Wolfsohn)	MAR 3 '23 MAR 29 1923
Glaser. M. F.	JAN 31 '24
Leland H. Studebaker	FEB 28 '24 NOV 30 '33
L. Rollin	APR 27 196